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DISEASES

OF THE

NERVOUS SYSTEM

BY

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PREFACE

The original purpose of this book was to furnish to the students under the writer's instruction a group of lectures upon nervous diseases which would be within their powers of comprehension, and at the same time, in moderate space, provide them not only with a somewhat dogmatic statement of neurological facts, but would also, so far as might be possible, give the reason for those statements. Having worked out this scheme, it has seemed that the general practitioner might be in a position to use such a collation to advantage, and so it is presented to the profession as an attempt to state existing facts more clearly, rather than to make any sensible addition to them. It has been the experience of the writer that books upon neurological subjects have fallen into two groups. The books of one class are so elaborate and technical that they confuse the student, since he cannot construct such an elaborate picture of a disease with definiteness enough to make it possible for him to carry it in his mind. The necessary bulk of complete exposition also blinds him to the fact that he has before him *in extenso* the pathological data, and the anatomical grounds, from which he should be able to work out the typical and the unusual symptoms which have been stated in detail. The second class of books avoid this danger by their brevity, but to attain this end they are obliged to state, with the greatest economy of words, the facts of a typical example only of the disease under discussion. Statements under such limitations of space are necessarily dogmatic, and the basis for the existence of the symptoms cannot be made clear. It has seemed to the writer that, in a branch of medicine where diseases are notoriously atypical, and tend to merge into one another to all degrees, the most useful text-book was one which would make as clear as possible the pathological basis of symptoms, wherever they might occur in the nervous system, and then the character of the atypical symptoms would at once reveal the pathological lesions which must be responsible for their appearance. This is of course the ideal, since we do not know the actual basis of all neurological phenomena, but it has been adhered to so far as has been found practicable. The medicinal treatment of many nervous diseases is at present considered to be futile, so far as cure is concerned, and the physicians of all schools are driven to symptomatological prescription, and to palliatives. Under such conditions, we have

a right to feel that we are the best symptom hunters yet evolved, and that by faithful work we may occasionally change the classical prognosis. For this reason the literature of our school has been diligently searched for remedies which are indicated, and also those giving some clinical basis for their employment. For the same reason the adjuvant and palliative agents, from all sources, have not been neglected, since the relief of suffering and deformity may be the only assistance which we can offer to many who come to us for relief. Under the conditions attending the compilation of this book, it was not feasible to denote the origin of data in the majority of instances, but the writer wishes to acknowledge his indebtedness to the many contributors of articles upon the several subjects in recent current literature, and especially to the authors of the current text-books on Diseases of the Nervous System, Therapeutics, and Electro-therapeutics. These have been freely drawn upon for material, but their presentation of the subject has been so often modified that the authors might not wish to be held responsible for the statements in their present form.

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616 Madison Ave.,
New York.

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DISEASES OF THE NERVOUS SYSTEM

CHAPTER I

THE ARCHITECTURE OF THE NERVOUS SYSTEM.

A detailed description of the brain and spinal cord is not properly the subject for description in a text-book of this character, but a presentation of some facts, with a different grouping and with less detail, is necessary in order that the significance of the symptomatology of nervous lesions may be correctly interpreted. In the center of the brain is a cavity which is enclosed by a wall of nervous tissue. This cavity is lined by a ciliated membrane, which is called the ependyma (garment), covering a layer of gray matter. This membrane is serous in character, and the cavity contains a watery fluid termed the cerebro-spinal fluid, which moderately fills it. This fluid is from 60 to 200 c.c. in quantity, increasing somewhat with age, has a specific gravity of 1.007, and contains about .01 of one per cent. of proteids, but no albumin. Its pressure is from 5 to 7.3 mm. of mercury, being about that of the blood in the capillaries. The primary division of this cavity-wall is into lobes, insula, frontal, parietal, occipital, temporal, and limbic. The outer surface of these primary divisions is moulded into roll-like masses to give more space for the cell-areas. This cellular layer is called the cortex. Internally the gray matter is in the form of laminae and nuclei, and extended into commissures to unite the hemispheres. These external mouldings are called gyri, or convolutions; the spaces between them are fissures, which are also called sulci.

The cell areas are gray in color, while the fibres connecting them are white. The cortex is gray therefore, and the central areas are white, being made up, in the greater part, of tracts of fibres joining different cell-areas. In various places we find areas of gray matter in the central white matter; nuclei, or ganglia. These are centers where nervous stimuli are changed in direction or character of impulse. These are the caudate nucleus, lenticular nucleus, claustrum, optic thalamus, corpora quadrigemina and geniculate bodies, and the anterior and posterior perforated spaces. While appearing separate they are connected with the gray matter of the cortex, and with each other.

communicates with the serous membranes outside of the brain by a small aperture in the roof of the fourth ventricle, called the metapore, or foramen of Magendie.

The brain is covered by three membranes; the dura externally, the pia internally, and the arachnoid lying between them. The two latter are sometimes considered as one, under the name of the arachno-pia. The dura has two layers, the outer being the periosteum of the skull, and through the foraminae of the skull is continuous with the pericranium. The inner layer (endocranium) is more vascular than the outer, its arteries being branches of the occipital, vertebral, ascending pharyngeal, internal maxil-

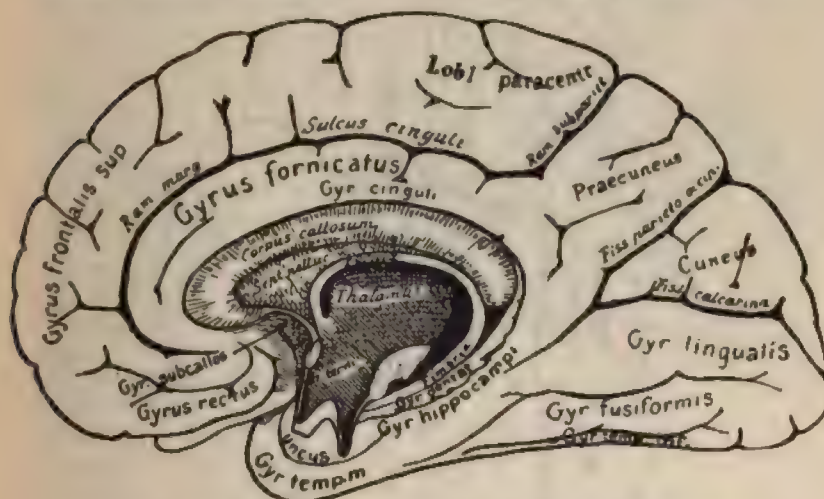


FIGURE 2.—MEDIAL SURFACE OF A HEMISPHERE OF THE BRAIN. (From Edinger.)

lary, internal carotid, and ophthalmic. It is covered upon its inner surface by a layer of epithelium, which is serous, and secretes a dram or two of serum, for the lubrication of the surfaces. This is not the cerebro-spinal fluid. This membrane develops projections which steady the brain in the skull. The greatest of these is the flax cerebri, which extends from the middle line of the vertex of the skull, and is attached to the crista galli of the ethmoid bone below, and the tentorium behind, and is the partition between the two hemispheres. The tentorium is a similar process which is arched over the cerebellum. Below is the vertical falx cerebelli, and in front is the diaphragm of the sella.

In forming these projections a triangular interval is left between the two layers of the dura, and thus are formed the sinuses which are lined by the intima of the veins which empty into them.

They are the receptacles of the venous blood from the brain, and empty it into the general venous system, anteriorly by the ophthalmic vein into the anterior facial, and posteriorly by the veins from the occiput into the external jugular. The blood from the ventricles passes out in the veins of Galen, which empty into the straight sinus, and thence into the occipital veins, and thence into the internal jugular.

The pia is spoken of as a thin membrane which covers the surface of the brain, and follows every irregularity of its surface,

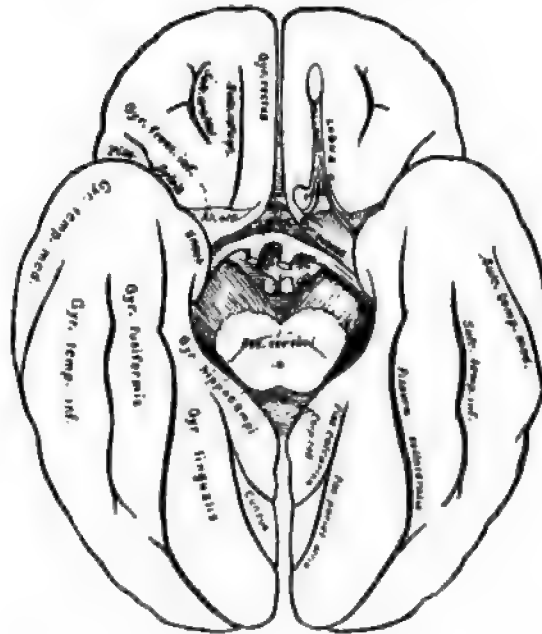


FIGURE 3.—CONVOLUTIONS AND FISSURES AT THE BASE OF THE BRAIN. (From Edinger.)

but we shall gain a better idea of its function if we conceive it to be a fine meshwork of blood-vessels and lymphatics, held together by a thin membrane. This blood-supply comes from the vertebrals and the internal carotid by way of the circle of Willis.

Lying between this pial membrane and the inner surface of the dura is a web-like tissue, the arachnoid (spider web). This extremely delicate structure differs from the pia in passing over the sulci of the brain instead of dipping into them, and also in sending off numerous trabeculae which divide the subarachnoid space into cavities of varying capacity. In these cavities lies that portion of the cerebro-spinal fluid which is found upon the

external surface of the brain, and in health the quantity is not more than a few drams. These spaces are very much more capacious in some localities, and are then called lacunae or cisternae. Two large ones exist at the base of the brain, forming a water-bed for the soft brain-mass. One of these is situated between the inferior surface of the cerebellum and the dorsal surface of the medulla, while the other extends in front of the medulla, pons, interpeduncular space, and crura cerebri to the optic chiasm, and laterally to the margins of the temporal lobes. This is continuous with the anterior subarachnoid space of the cord, and above with the smaller spaces developed in front of the optic chiasm, one in each of the fossae of Sylvius, a space over the corpus callosum, and the spaces of the medulla and cerebellum.

The blood supply to the brain comes from the internal carotid, and the vertebrals which arise, usually, from the subclavian. The perfection and certainty of supply are conserved by means of the circle of Willis. This is made up by a posterior artery of origin, which is the basilar, formed by the union of the two vertebrals. The

basilar divides into the posterior cerebrals; they each send forward a prolongation, the posterior communicating, which unites with the internal carotid; the internal carotid sends forward the anterior cerebral, and the two anterior cerebrals, by their union in the anterior communicating artery, complete the circle. From this circle all portions of the brain, both external and internal, derive their arterial blood. An upward extension of the internal carotid furnishes the greater part of the blood supply to the brain; this is the middle cerebral which runs in the



FIGURE 4.—CIRCLE OF WILLIS.

A. Internal carotids. B. Ophthalmic. C. Posterior communicating. D. Anterior cerebral. E. Anterior communicating. F. Middle cerebral. G. Lacrymal. H. Short ciliary. J. Central retinal. K. Muscular. L. Frontal and nasal. M. Vertebrals. N. Posterior dorsal. O. Posterior spinal. P. Anterior spinal. Q. Inferior cerebellar. R. Basilar. S. Internal auditory. T. Superior cerebellar. U. Posterior cerebral. (Modified from Bourguery.)

Sylvian fissure, and gives off the ganglionic branches to the interior of the brain. The details of supply are as follows: The vertebrals, after giving off the anterior artery of the spinal cord, and the posterior spinal arteries, give rise to the posterior cerebellar arteries, while the anterior inferior and the superior arise from the basilar. The posterior cerebral nourishes the internal surface of the occipital lobe, the under surface of this, and the temporal also, and a small portion of the outer aspect of the temporal. The anterior cerebral is distributed to the median surface of the hemisphere back to the field of the posterior cerebral on the occipital lobe, the anterior part of the outer surface of the frontal lobe, and a small strip on the external aspect of the vertex clear back to the occipital lobe. The middle cerebral

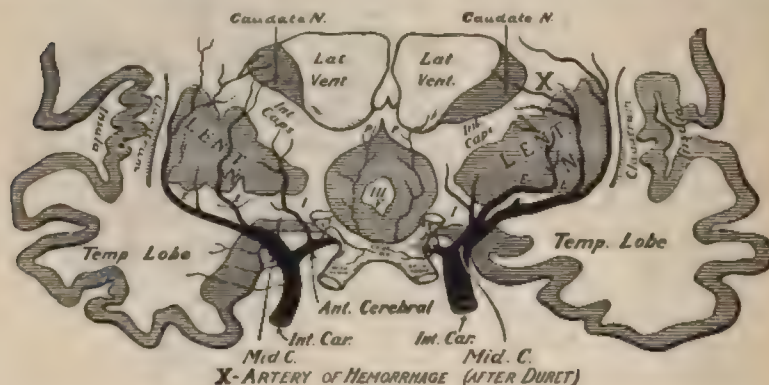


FIGURE 5. — TRANSVERSE SECTION OF BRAIN.

These are the Lenticulo-striate arteries and are the most subject to rupture, especially upon the *left* side. Since they supply the region of the Internal Capsule their rupture naturally produces a hemiplegia, paralyzing motion and sensation on the opposite side of the body.

supplies all the rest of the cortex, and sends off the lenticulo-striate, and lenticulo-optic branches. The former nourishes the lenticular and caudate nuclei, except the head of the latter, while the last named supplies the internal capsule, and the anterior and inner parts of the optic thalamus. The largest of the lenticulo-striate branches passes between the lenticular nucleus and the internal capsule, and is the "artery of hemorrhage" of Charcot. The finer distribution of the blood supply is principally important when it relates to the cortex. This is nourished by the blood-vessels running in the pia, from which its distribution is effected by a long and a short set of branches which do not communicate, and which branches are terminal, in the sense that they have such a slight degree of anastomosis that an area of softening will result from their obliteration. The short arterial twigs go to the

gray matter, while the longer ones nourish the underlying white substance. The ganglia and nuclei have a similar supply.

The veins of the brain are peculiar in having no valves, in possessing a very thin wall (having no muscular coat), in emptying into the sinuses, in frequent anastomosis, in being less in number, but greater in capacity than the arteries, and in that the superior ones empty into the superior longitudinal sinus, against gravity, and against the direction of the flow in the sinus. It is to be noted here that thrombosis in the veins is made very probable from the facts just mentioned, and in the sinuses from the fact that these latter are intersected by trabeculae. Another fact in relation to the lumen of the sinuses is that projections of the pia through minute openings in the inner wall of the dura encroach upon the lumen of the sinuses and drain off the cerebro-spinal fluid under some conditions. These are called Pacchionian bodies, and while projecting most usually into the superior longitudinal sinus, invade other sinuses as well, and may indent the bones of the skull. The veins are divided into the superficial and deep. The superficial are the anterior and middle cerebral, and the basilar, the latter being formed by the junction of the two former. The anterior cerebral drains the median surface of the frontal lobe, and the convex surface of the corpus callosum, and unites with the middle cerebral. The middle cerebral covers a less space than the artery of the same name, lies in the fissure of Sylvius, and overlies the island of Reil, and receives the blood from this area, and unites with the anterior cerebral. The basilar runs backward from its point of origin across the crus, and around the corpora quadrigemina, and uniting with its fellow of the opposite side, and the veins of Galen from the ventricles, forms the straight sinus. The union of the two basilars in the rear, and the two anterior cerebrals in front, complete a venous circle similar to the arterial circle of Willis. The veins from the convolutions empty into the sinuses or larger veins. The deep cerebral veins drain the interior parts of the brain, and empty into the veins of Galen, which unite with the two basilars to form the straight sinus. The cerebellum has a superior and inferior system of cerebellar veins. The former empties into the veins of Galen, or the straight sinus, and the inferior into the petrosal and lateral, or the occipital sinuses.

Below the posterior part of the brain, under the tentorium, lies the cerebellum in the posterior fossa of the skull. It has no cavity, but is composed of three parts. The vermis, which is small, but by far the most important, and a lateral lobe on either side. The whole organ is made up of little leaves, and imbedded in each lateral lobe is a nucleus of irregular outline which is white within, but gray without, except at one point. The cerebellum, like the brain, has an exterior, or cortex, of gray matter, and a white

central portion, striated by secondary and tertiary layers of gray matter.

Just under the cerebellum lies the medulla, which is the posterior projection of the brain, the development of the fifth vesicle of embryonic life, and downward from which extends the spinal cord.

ANATOMY OF THE SPINAL CORD.

The cord is made up of thirty-one or two segments, which are the sources of the same number of pairs of spinal nerves, which are mixed in function, being composed of an anterior bundle which is motor, and a posterior which is sensory in function. These spinal nerves arise from the lateral margins of the segments. The segments are independent, each being the source of a single pair of spinal nerves, but are interdependent, since two or three segments are associated in the performance of any muscular act, or sense-perception, and the requisite coördination is ensured by the linking of the several segments to one or more, both above and below, by tracts of white fibres which run in what are called the anterior, or antero-lateral ground-bundles, association-tracts. The cord is about eighteen inches in length, weighs nearly an ounce, and extends from the lower border of the atlas to the upper border of the second lumbar vertebra, i.e., to about the level of the inferior border of the scapulae. The spinal nerves are level with the spinous processes in their origin and exit from the spinal canal at the upper part, but in the upper dorsal the spinous processes begin to trend downward, until in the lower dorsal we must look for the width of a vertebra, or one and a half vertebrae, above, for the point of its origin in the cord. Below the ending of the cord in the conus terminale, the nerves of the lower lumbar, sacral, and coccygeal segments in their long course to their points of exit, are gathered into a compact bundle called the "horse's tail" (*cauda equina*). From the fifth cervical to the second dorsal there is an enlargement, in order to give space for the development of the cell-areas necessary to innervate the upper extremities, and a similar, but smaller and shorter one, from the tenth dorsal to the second lumbar for the service of the lower extremities.

The cord, like the brain, is surrounded by two meningeal envelopes with the arachnoid membrane lying between them. The meninges are similar in function to those of the brain, but with some differences which have a physiological and pathological bearing. The dura of the cranium is applied directly to the inner surface of the bone, while that of the cord is separated from that of the vertebrae by a cellular structure, rich in fat, and ramified by vessels and lymphatics, rendering epidural hemorrhage much

less dependent upon external violence, than in the case of the cranial dura. The pia is just as closely applied to the cord as is the pia to the brain, but differs in the fact that the pia of the cord ramifies its substance with numerous septa, the folds of which form lymphatic spaces, and thus allows infections a ready entrance to all parts of the structure.

Between the dura and pia is the arachnoid, having a slight space filled with fluid between it and the dura, subdural space, and a much greater one between it and the pia, subarachnoid space. The latter by way of the metapore, (an orifice in the membrane over the medulla), connects with the subarachnoid space of the brain, and thence with the cavities of the ventricles, making all these cerebro-spinal spaces into one, filled with what we know as the cerebro-spinal fluid.

The cord is sustained in its position vertically by the attachment of the membranes about the margin of the foramen magnum at the top, and by the fusion of the filum terminale with the periosteum of the coccyx, or of the posterior surface of the sacral canal. The filum is a fine cord made up of all the membranes fusing together below the conus. Lateral motion in the cerebro-spinal fluid is prevented by the ligamentum denticulatum. This is a lateral band of fibrous tissue passing from the pia to the dura, running peripherally from the sides of the cord, and thus forming a partition which divides the subarachnoid space into a posterior and an anterior part. It is united to the periosteum of the vertebrae by about twenty digitations which are attached to the bodies of the vertebrae between the intervertebral foraminae.

The spinal nerves pass from the segments in an anterior and posterior bundle (actually the anteriors pass out, while the posteriors are on their way inward) on the outside of the dura, except in the case of the sacral nerves, and on the pedicle of the vertebrae there is a ganglion containing the nutrient cells of the posterior root-fibres which is called the posterior root-ganglion. Just outside of this ganglion the two bundles come together, surrounded by an outer envelope composed of the dura and pia from the cord, and inside the pial envelope is the perineural lymph-space communicating with those of the pia in the substance of the cord, and the lymph-spaces in the epidural area.

The circulation of the cord is provided by one anterior and two posterior arteries. The anterior arises from two branches from the vertebrae within the skull. The two posterior, from the same source of origin, run about the posterior aspect of the medulla and gain the posterior aspect of the cord, just anterior to the posterior roots. The arteries in the substance of the cord are terminal as in the brain. The anterior artery runs in the anterior fissure the whole length of the cord, and receives numerous accessions from the branches of the intercostal, lumbar and sacral

arteries, which pass in with the nerves from these localities. The method of supply from the anterior spinal artery is peculiar. The artery gives off about three hundred primary branches at right angles to the parent stem. These branches do not divide to supply both sides at one level, but a branch goes only to a right or left anterior horn. This branch to this half divides, one part going to nourish the anterior horn, and the other to the neck of the horn, and to partially supply the space about the central canal, and the anterior part of the posterior part of the cord. From the primary branch another division is given off vertically to unite with the branch above it.

The two posterior arteries lie just anterior to the posterior roots, and divide into branches which nourish the posterior structures of the cord, and partially the central also. The posterior arteries anastomose on the periphery of the cord with each other, and also with the anterior arteries. The cord therefore has an area nourished exclusively by the anterior arteries, another by the posterior, and a third which draws its supplies from both, and is therefore more immune than the brain from the effects of an occlusion of a single artery, or set of arteries. Owing to the separate supply to the right and left halves of the anterior horn, the occlusion of a single branch can affect only one half of an anterior horn, so that a limited lesion may produce a disablement of one side of the body only.

The white tracts of the cord are nourished by superficial branches from the periphery.

The vascular system of the cord is peculiar again in the fact that it has the longest arteries of their size in the body, and that they receive the blood at a low pressure, and that the veins have to act in opposition to gravity. If, therefore, the condition of the arteries is such that their elasticity is reduced, an ischemia is very likely to occur, and softening is produced. The anterior part of the cord has an abundant arterial supply, and a poorly developed venous system, so that pressure is high, and rupture is likely to occur under proper conditions, while just the opposite is true of the posterior part of the cord.

There is no true lymphatic system in the brain and cord, but the spaces between the pial envelope of the vessels and the brain tissue (circumvascular spaces) and the fissures produced by the infolding of the pia in the substance of the cord are lymph channels, and connecting with the general lymphatic system fulfil the same function. They are called the spaces of His. Since the flow from the periphery of the cord is lowest just as the perivascular lymph-spaces open into the spinal canal, the spinal roots are kept longer in contact with a possibly infected lymph than are any other structures. This is probably the method of toxic infections of the cord.

HISTOLOGY.

The histological elements of the nerve tissue are Nerve-cells, Nerve-fibres, End-organs Neuroglia-tissue, Blood-vessels, and Lymph-channels and Spaces.

The nerve unit is the neuron or neurodendron, and is made up of three parts.

1st. The nerve cell-body or neurocyte (neuron, a nerve, and kutos, a cell).

2d. The protoplasmic processes of Deiter, called also the dendrites of His, with their terminal ramifications.

3d. The so-called axis-cylinder process, with its collateral branches, and terminal end-brushes, called telodendrons.

The nerve cells, or neurocytes are arranged in distinct groups or layers in different localities, and the areas have various shapes. In the brain there is a layer over the whole exterior aspect of the brain, and lining its cavities, while in the deep portion of the brain the cells are in small masses of various contour. In the hind-brain we find other collections, and in the cerebellum they occur like thin plates of various shape in the leaves of white tissue which make up the organ. In the crura we find the cells in narrow streaks, which continue into the pons, where again we encounter small masses. The medulla has gray matter in vertical streaks, and when we examine the cord we find that these streaks, broadened out into somewhat butterfly-like contour, have such a long vertical extent that the name "column" has been given to them. Wherever in the central nervous system we find the whitish appearance changed to a gray, we may safely assume that we are looking at a collection of cells, and if the area is a small one it is called a nucleus, but if larger and somewhat greater in bulk it is apt to be termed a ganglion, while if of long vertical extent it will be termed a column.

These cells present a variety in contour and size, the most general one being irregular, large or small, multipolar forms with dendrites, or protoplasmic processes, varying from one to seven in number. These arise from irregularities in the surface of the cell-body, branch like a tree, but do not anastomose with one another, nor with the dendrites of other cells. They vary in length and thickness, and in their course develop frequent varicose swellings, and on their sides are minute lateral bud-like projections called gemmules. The function of the cell-body is considered to be nutritional, while that of the dendrites is in some way to establish contact with other cells by the way of similar dendrites given off by them. The method by which this interchange of nervous impulse is accomplished is a matter of theory, some holding that

an energized cell imparts to its dendrite an erectile quality by which it is brought into physical contact with the dendrite of another cell, much as when we make an electrical contact in a bell-circuit by pushing in a button.

The typical cell is a granular protoplasmic body containing a large, usually centrally placed, nucleus, containing one or more nucleoli. This nucleus is irregular in form in the living tissue, but after death assumes a circular form. It is surrounded by a delicate membrane, and consists of a network called, from its affinity for stains, chromo-plasm, and in its meshes is a more

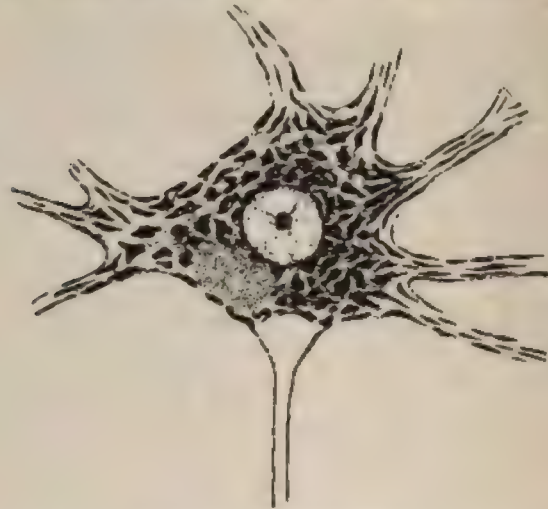


FIGURE 6.—LARGE MOTOR CELL.

From the anterior horn of an ox. Highly magnified. The axis-cylinder process, or neuraxon, is the downward prolongation. The others are dendrites. This cut shows the nucleus, and the arrangement of the chromophilic granules which are concerned in the nutrition of the cell. (From O'Connor.)

fluid material called karyoplasm. Many of the cells of the sympathetic system contain two or more nuclei. The nerve cells of the central nervous system have no enveloping membrane, but simply a condensation of tissue. About the nucleus of the cell, protoplasm exists in two portions; one is stainable to an intense degree, and the other a delicate coagulable substance of unknown character, which will not stain. When the stainable portion is arranged in stripes, the cell is called stichochrome, but when it is in a network the cell is termed arkyochrome. There are two kinds of stainable matter in a cell. The first is granular matter in an amorphous cement-like substance which is found in definite small collections, which will stain deeply, and are called Nissl

bodies. The other stainable substance is heaped at the base of a cell in a unipolar cell, and in any case about the point of exit of the axon. In health this is a pale yellow, but in case of degeneration of the cell it stains a deep black with osmic acid, and also is found to have changed into a sort of powdery mass in place of being in granules. The non-stainable portion of a cell consists of fibrillae, which pass in a network between the Nissl bodies, and pass into the dendrites and axis cylinders. The ordinary forms of cells are bipolar, multipolar, unipolar, pyramidal, Purkinje, and basket.

Bipolar cells are found in the ganglia of the sympathetic system, posterior spinal ganglia, ganglia of the sensory cranial nerves (in embryonic life), in the molecular layer of the cortex (outside layer). They are spindle or pyriform in shape, and have a fine single axon springing from each pole.

Multipolar cells, are the largest of all in the nervous system, varying from 8 to 120 micromillimetres in diameter, which makes the largest sixteen times the size of a red blood-corpuscle, are found throughout the entire nervous system, but predominate in the anterior horns of the spinal cord, the medulla, the cerebro-spinal ganglia, and the peripheral ganglia of the sympathetic.

Purkinje's cells are found in the cortex of the cerebellum, in a distinct layer, lying between the external or molecular one, and the internal or granular layer.

Basket cells are peculiar to the cortex of the cerebellum, and they throw off axons having a horizontal course, continually giving off descending branches (collaterals) which terminate in brushes about the bodies of the cells of Purkinje. The name is evident when we see that the cells of Purkinje lie in a mass of the bushy collaterals as in a basket.

Pyramidal cells are multipolar, triangular, or pyramidal in shape, and possess a fine dendrite at the apex which tapers as it approaches the superficial layer of the cortex. Delicate dendrites project from all sides of the cell body, while from each corner of the base springs a dendrite, which extends obliquely to the plane of the vertical fibres of the cortex, and there divides into numerous branching filaments studded with gemmules. The axis-cylinder process usually springs from the middle of the basal portion of the cell-body, passes vertically downward, has great length usually, is smooth, less in diameter than the dendrites, and gives off right-angled collaterals. In most cases axons receive, soon after leaving the cell-body, a layer of myelin and become medullated nerve-fibres. Such medullated fibres constitute the white substance of nervous tissue throughout the nervous system, and always mean nervous paths, as opposed to cells, where some changes of a chemical nature take place upon which nervous impulses hang in some way. Stated in a dogmatic way, axons

conduct sensory impulses to, and motor impulses from, cell-bodies. All peripheral axons end in: (1st) inter-epithelial arborizations; (2d) specialized end-organs; (3d) motorial end-plates. The first two modes of termination appertain to sensory nerves only, while the latter refers to the combined sensory-motor nervous apparatus in voluntary muscles.

There are six chief forms of specialized sensory end-organs: (1st) The tactile corpuscles of Meissner and Wagner; (2d) the end-bulbs of Krause; (3d) Pacinian or Vater's corpuscles; (4th) tactile menisques; (5th) corpuscles of Golgi; (6th) so-called muscle-spindles. A large number of the sensory nerves do not end in these specialized methods, but in the inter-epithelial arborizations referred to before. In this event the axon as it approaches the skin, or the surface of the mucous membrane, loses its myelin and neurilemma, and the axis-cylinder, thus left naked, repeatedly divides into a plexus of fine fibres, which terminate in the epithelial layers of the given tissue, or the layers of other structures which it supplies. This method of termination is true of the skin, mucous membrane, hair-bulbs, teeth, tendons, the muscles and many of the glands.

Among the specialized end-organs we find the tactile corpuscles of Meissner and Wagner where touch is most developed, viz., in the ends of the fingers and toes, less in the tip of the tongue, skin, lips, etc. These corpuscles are oval in shape, 1-3000th to 1-800th of an inch in length, and about 1-800th of an inch in thickness, composed of a spiral of nerve-filaments about, and ramifying, a mass of connective tissue.

The end-bulbs of Krause are small cylindrical or oval corpuscles, slightly bent at their base, made up of nucleated connective-tissue capsules, in which there is a core of soft protoplasm, containing numerous cells, upon which the naked axis-cylinders rest. These are found in the mucous membrane of the lips, nose, mouth, conjunctiva, in the papillae of various extremely sensitive mucous membranes, and in the synovial membranes of the joints of the fingers, where they are called articular end-bulbs.

The Pacinian or Vater corpuscles are irregular oval-shaped bodies, 1-20th to 1-10th of an inch in length, situated on some of the cerebro-spinal sympathetic nerves, composed of laminated connective-tissue capsules, with 40 to 50 laminae like an onion, with a soft protoplasmic core, through which the axis-cylinder, bereft of its myelin sheath, passes to nearly the extremity of the capsule. Here it ends in a sort of varicose swelling. These are found in the subcutaneous tissue on the nerves of the fingers and toes, near the joints, on the intercostal nerves, those of the arms, neck, nipple, external genitalia, and on the nerves of the abdominal sympathetic, and the mesentery.

The tactile menisques of Ranvier occur in both the superficial

and deep layers of the skin, and are plexuses of nerve-fibres forming arborizations whose branches become flattened so as to resemble leaves.

The corpuscles of Golgi occur in tendons near their point of insertion into the muscles. The tendon-fibres become somewhat enlarged, and the medullated fibres, after losing their myelin, penetrate these fibres; then as an arborization break up into a number of fibrils, which form terminals somewhat spindle-shaped. The enlargement of these fibres of the tendons, together with these terminal arborizations of the nerves together form the structures which are known as the corpuscles of Golgi.

Muscle-spindles are found in nearly all the skeletal muscles, but are especially abundant in the biceps of the arm, and the small muscles of the hand. They are more in the belly of the muscle than in the tendons, uncommon in the muscles of the eye, diaphragm, and the intrinsic muscles of the tongue. They consist of muscle-fibre, nerve, their endings, and a connective-tissue sheath, blood-vessels and lymphatics. The sheath is composed of from eight to ten laminae. Its function is the setting up of contractions in muscles. They are found degenerated in tabes.

Motor nerves terminate in both voluntary, and involuntary muscles. In the voluntary muscles the nerves are all medullated, and have their origin in the cerebro-spinal system. The nerves to the involuntary muscles are non-medullated, and belong mostly to the sympathetic system. Motor nerves to voluntary muscles terminate chiefly in special expansions called motor nerve-plates or organs, motor-sprays, fields of innervation of Kuhne, eminences of Doyers, or sarcoglia. These end-plates are located below the sarcolemma of primitive muscle-fibre, are continuous with their sarcous substance, are flattened or slightly elevated masses of granular protoplasm, irregular, spherical, or oblong in shape, contain cells with an investing envelope, and clear nuclei, and nucleoli. Each motor-fibre passing into a muscle repeatedly divides at the nodes of Ranvier into branches both ascending and descending. These subdivide into a number of branchlets, each pursuing an oblique or transverse course between muscle-fibres forming an intra-muscular nerve-plexus. Each primitive nerve-fibril, still medullated, passes to a muscle-fibre, having divided just before reaching it. As it enters it, it loses its myelin, and neurilemma, and becomes continuous with the sarcolemma of the muscle-fibre. The axis-cylinder passes on under the sarcolemma, resting on the motorial end-plate, where it divides into two or three primary branches, which further subdivide into ultimate flat twigs which expand at their ends into minute bulbs. The nerve-termination is therefore a distinct non-anastomosing arborization, the whole resting upon a motorial end-plate, which is in turn continuous with the sarcous element of the muscle-fibre, and

it is probably the cause of the contractile wave of the muscle, as its point of origin is in the motorial end-plate.

The motor nerves for involuntary muscles are non-medullated, and come mostly from the sympathetic nervous system. Near their termini they ramify, and form a close network, or plexus of fibres. In the angles formed by the crossing of the fine fibrillae of this network, ganglion cells are found. From these ganglionic plexuses fibrillae arise which pass between the muscle-fibres, and continue parallel with them, frequently bifurcating in their course, each division giving off small branches which terminate in varicose or bulbous extremities opposite the nuclei of muscle-cells, without passing into them. According to Arnold and others, they do pass into the muscle-cells and end in their nuclei.

The brain and the other parts of the central nervous system, i.e., the brain, bulb, and cord, are essentially simple aggregates of the cells which have been described, and their prolongations into dendrites, or axons. As in all other structures of the body, however, a framework of neutral tissue is found, which here is called neuroglia, or simply glia. This is a substance made up of cells with many hair-like filaments, which are termed glia, or spider, cells. They are in a state of potential overgrowth, so that when true nervous tissue becomes disintegrated, the glia cells spring into accelerated growth, and fill in the space left by the decay of nervous tissue. It is like scar-tissue in other parts of the body, and is the reason why organic diseases of the nervous system are so often incurable. The glia tissue has filled in the space, and no renewal of nervous tissue is possible.

PHYSIOLOGY.

PHYSIOLOGY. In order to appreciate the causation of diseases of the nervous system, we must grasp the fact that each nerve-unit (neuron-cell, dendrites, axon, terminal brushes) is an easily excitable body, and that all movement and function may properly be considered as a response to peripheral or central stimuli. If the spinal centers or their axons are injured, the result will be disease of the cord, as a reflex organ. If the brain or fibres from it in the cord (pyramidal fibres) are invaded, there will be a loss of the power of inaugurating movements in response to the will, of intellectually appreciating the significance of external stimuli, and of inhibiting the reflex action of the cord. If the terminal brush of a nerve-fibre is applied to non-nervous tissue, the axon is a motor one, and is efferent, or one conducting impulses from the center to the periphery, while the contrary is true if the terminal brush of an axon ends about nervous tissue. One end of each neuron is receptive, and the other emissive, and the cell is

the nutrient center for the whole structure. The connection between terminal brushes and cells is made by contact of the terminal brush and the recipient dendrite by erectility, or through the agency of an amorphous substance lying between them, the whole mechanism being then called a synapse. The function of the synapse is conjectural, and it may be a substance capable of allowing nervous impulse to pass only under certain conditions, which would not militate against a theory of continuity of nerve-fibre, or it may be a body in which the actual contact of the fibres takes place after erectility had been produced by a stimulus to the terminal. They may be capable of this erectility only when in a condition of health, and this is possibly the cause of the lack of potential energy, both mental and physical, of a tired person. Nervous energy is exceedingly slight in amount, and traverses a fibre in one direction only (perhaps impulse is made uni-direction by the synapse), and at a rate of thirty metres per second. As a wave of nervous energy flows along a nerve it does not influence similar fibres, or the structures near it, but at a synapse it stimulates whatever dendrites are imbedded in that structure, and therefore at that point it passes the impulse on to other fibres, and at that point, and at no other, may change the direction of the impulse, and also diffuse it, since one impulse may here be received by many dendrites relating to different cells. This is the law of isolated conduction of J. Müller. This diffusion depends upon the fact that every cell has numerous dendrites, and that the terminals of a cell are in contact with numerous muscle-cells. Each neuron ends in as many as thirty brushes, and a small muscle may be innervated by two hundred spinal neurons. A simple reflex is made up of a short line with few neurons and synapses; this is a simple reflex with impulses in a single direction, whereas a more complicated arrangement means many alternative directions; this latter is the reflex used in habitual movements, and specialized groups of movements, as those of the musician, and lastly, when fibres from the ideational centers are introduced, we have the highest type of nervous linkage, where operations are carried on either reflexly or by volition.

TONUS. Every structure of the body is kept in a state of functional readiness by a constant flow of rhythmic impulses (twelve to sixteen per second), the cortical cells toning up the motor cells of the cord on the opposite side of the body, while the cerebellum energizes those of the same side. The higher cells are impelled to this by the constant flow of sensory stimuli from the periphery. When a movement is inaugurated, the tonus of the cell is suddenly pushed up to the point of discharge by an augmentation of the force of this rhythmical impulse. Nervous stimulation becomes motion or function by setting off a chemical discharge in the structures. Myotatic irritability is an increased

readiness of chemical combination, but the mechanism is not certain.

TREMOR. All movements which we make are slightly tremulous, for the simplest possible movement consists of the fusion of at least five contractions per second, and a movement can be repeated about eleven times per second. When the cells of the motor cortex, or the pyramidal tracts, are irritated, we may have irritation-contraction, but, although this seems to be a constant tension spasm, actually it is slightly tremulous. In general, tremor may be considered an evidence of a degree of loss of nervous energy, which, if further developed, will end in paralysis. It may arise from a disease of the cortical cells, as perhaps in alcoholism, or general paresis, or from interference with the descending tracts from those cells, either in continuity, or in the synapses, or to an increase of the inhibitory action of the higher motor-cells upon the lower. Tremor does not arise from a degeneration of pure muscle, but in cases indicating such an origin there has always been a previous injury to cells. In normal old age we see it only in voluntary movement, while if the tremor is of a part at rest, it shows an abnormal degree of cell-degeneration. The tremor of paralysis agitans arises from the fact that the impulses are too slow for fusion, and it is probably the result of a pre-senile change in nerve-cells, or in the synapse. The cells of the cortex (through the fibres of the direct pyramidal tract) inhibit a too ready motor discharge of the spinal motor-cells in response to peripheral stimuli, and certain nerves inhibit certain muscles (as the vagus does the heart), and certain regions of the brain inhibit definite parts of the body. A moderate increase of inhibition (from a general psychical stimulant like fear) will produce tremor, but if more intense will result in paralysis. Chorea may produce its characteristic incoördination in this way. Disease of nerve-fibre does not generally produce tremor, but some degree of paralysis, and when tremor appears to arise from such a cause it is probably due to degeneration in the synapse. Disseminated sclerosis is a disease marked by tremor, and probably from this cause, and in general paresis the same condition is present in the cortex, and it occurs in cerebellar disease, but only when the middle lobe is affected. It is either intention (evident only on, or exaggerated by, volitional movement), or passive, when it is only present when the part is at rest, and is stilled by attempts at voluntary motion (paralysis agitans is a typical form, being constant except in sleep).

REFLEX ACTION. All that is essential to a reflex is a sensory nerve to appreciate stimuli at the periphery, the connection of the recipient cell with a motor-cell, and a fibre from the cell to the mechanism at the periphery through which this motor-cell discharge may be translated into action. The function of the

sympathetic ganglion, the cerebellum, and the brain is to hold this reflex excitability under such a degree of control (inhibition) that the organism shall not be in such a state of unregulated

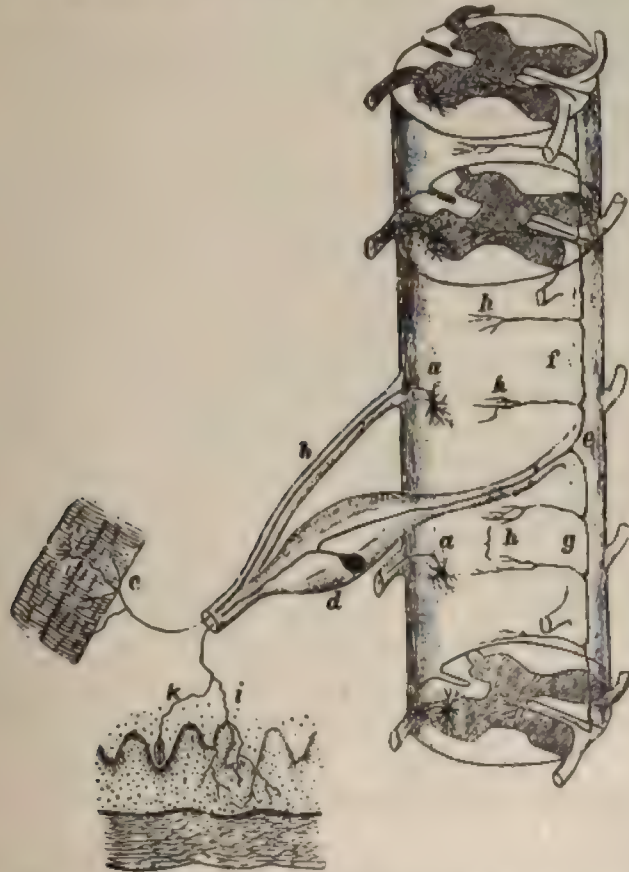


FIGURE 7.—SCHEME OF A SPINAL REFLEX.

Cross sections of the cord are shown with the projecting nerve-roots; *c* is a motor nerve, ending in a muscle; *k* and *i* sensory terminals in the skin and tissues; *k* is a Meissner's corpuscle, and *i* a naked arborization. They unite in the spinal nerve *d*, being the ganglion on the sensory root. *b* the motor fibre originates in a motor cell in the anterior horn, while *d*, the sensory fibre ends in a sensory cell; *h*, the dendrite shown at *h* arborizes about one of the cells of the posterior horn. From this a new axon is sent across to arborize about one of the motor cells *a*, and thus the reflex arc is completed. (From Lenhossek.)

response to stimuli continually being brought in, that its energies will be exhausted in futile reactions. The reflexes which are most evident to us in health and disease are tendon reflexes, skin

reflexes, and the automatic functions of the bladder and rectum. It will readily be seen that if the reflex arc is broken in any part the reflex will be diminished or abolished, or if the flow of tonus from the brain is absolutely annulled, as in coma, or if inhibition is greatly increased, as in hysteria. The reflexes will be increased, on the other hand, by any condition which diminishes or cuts off inhibition, as hemiplegia, or in the functional neuroses, which increase the excitability of nerve-cells, or by degenerations of the pyramidal tracts.

TENDON-REFLEXES. These are commonly attributed to the propagation of a sensory stimulus by a blow, upon the patellar tendon for instance, which sends in through the sensory fibre a stimulant which irritates and discharges a motor-cell in the anterior horn of the cord, which in turn produces a contraction of the quadriceps extensor muscle. It is known to be absent in cases where there is destructive disease of the muscle, the sensory nerve, the motor nerve, or the spinal center. In spite of these lesions being sufficient to abolish this so-called reflex, it is not actually a reflex: This is proven from the fact that the reaction time is too short (.008 of a second, while nervous impulse is thirty metres per second, if it encounters no synapse) to enable the nervous impulse to travel the necessary distance. The reason that the integrity of the spinal centers and conduction-tracts is necessary is because the tonus of the muscle depends upon their condition. The reaction is a simple spasm of the quadriceps extensor varying in amplitude according as this tonus is depressed, normal, or exaggerated. Knee-jerk is always found in health, (some say absent in one per cent.), and should be single, and not reduplicated. If a stimulus is very pronounced, it may set up similar reactions in the opposite limb, while if still higher in degree the upper limbs may also be affected. This is due to the coördination of different segments by the association tracts. Other tendon-reflexes are similar. Every muscle has probably a similar jerk, but the elbow, wrist, jaw, and tendo Achillis are accessible, and so are the ones generally examined.

BLADDER AND RECTAL CONTROL. The reflex centers for these organic reflexes are located in the fourth and fifth sacral segments, that of the bladder being below that of the rectum. The sensory stimuli arise in the mucous membrane, and the motor impulse permitting evacuation is two-fold, the tonic contraction of the sphincters is inhibited, and a contraction of the muscles of the organ is inaugurated. Owing to the fact that the somewhat complex process is under the coördinating guidance of a sympathetic ganglion the act is a very perfect reflex, and avails to empty the bladder at proper intervals, even if the path to the brain is cut off, as in the case of dorsal myelitis, but the rectum fails to functionate under the same conditions. This condition of the

bladder is called active incontinence. If the sacral centers are destroyed the reflex is of course annulled, and the over-filled bladder will force open a weak or normal sphincter, and produce a dribbling discharge of a more or less constant character, this being termed passive incontinence. If the sphincter is in a state of tonic contraction, rupture of the bladder is likely to ensue. There may be a weakness of the sphincter which allows dribbling, or retention may take place from weakness of the detrusor.

CLONUS is the condition where the single response of a tendon-jerk is reduplicated so long as the muscle is kept in an over-extended position, and indicates a greater degree of loss of inhibition. This is never found in functional disease, and can only arise from a degeneration of the pyramidal tracts to a greater degree than is necessary to produce a simple exaggeration of the tendon-reflexes. If the second and third lumbar segments are destroyed, and the pyramidal tracts are degenerated, knee-jerk will be lost, but clonus will be present.

SPASM is a contraction of the muscles exceeding the limits of health, both in intensity and duration, without volitional stimulation, or when such stimulation produces a contraction of inordinate duration. Some cases arise from a simple paralysis of opposing muscles, or some intoxicant in the blood may set up an inflammation of the fibrous sheath of the muscle, or the muscle-fibres, which, in turn, increases the irritability of the nerve-centers, but in general we may feel sure that the centers of nerve-energy are irritated, or while inhibition is weakened as one factor, the same influence has simultaneously debilitated the cells in the cord, and has made them too susceptible to peripheral stimulation.

TONIC SPASM indicates that inhibition has been weakened; if the degree is a little greater we find clonus; and if still greater, there is contracture. This is when the position assumed during spasm is retained permanently.

CLONIC SPASM is the condition when the contraction alternates for a period with relaxation, at a rate which is uniform for a single attack, but may vary in successive attacks of the same individual, or in the attacks of several individuals.

TONIC SPASM can be proved to sometimes depend upon a cutting off of inhibition, since the knee-jerk will return to the tabetic if he suffers from a succeeding lesion which cuts off the pyramidal tracts, and it will occur in a tumor of the cerebellum if the growth compresses the pyramidal tracts. The cause of clonic spasm is not so certain, but conditions which produce a hemiplegia are competent for its existence. If a muscle remains for months in a state of contraction, changes arise in its structure, and contracture is likely to result.

SPASM may occur in peripheral nervous diseases where the

motor-fibres are irritated, but it is not universal in these conditions, and is not usually a marked symptom. In diseases of the sensory nerves it is more common to find spasm, but it is not proven that it is not the result of the debilitated condition of the spinal centers. In tetanus all the centers suffer from the intoxication, and therefore a slight peripheral stimulation tends to a sudden release of nervous stimulation to the muscles.

REFLEX SPASM sometimes occurs, but mostly in children, and in them the pyramidal tracts are poorly developed, and here it may also be based upon deficiency of inhibitory action. When a muscle is put upon a stretch by its opponents, it contracts so as to steady them. If this is continued for a length of time, the centers may fall into a state of irritable exhaustion, and spasm of the overstretched muscle may occur. Functional spasms may arise without evident disease, and while they are spoken of as functional, they probably arise from changes in the cortical cells, which result in a weakening, again, of inhibition. It is worth noting that, although they exhibit themselves as unilateral, they will be found, on close investigation, to be bilateral, and to be accompanied by more or less evident weakening of strength and of the senses, especially evident on the side of most evident spasm.

SPASM of involuntary muscles is due to irritation from local causes when in the digestive tract, and sometimes in the circulatory system, but is also a result of cortical changes.

INVOLUNTARY TWITCHINGS arise from irritation of the motor cells when the condition is not severe enough, nor sufficiently widespread to set up a contraction of the whole muscle. The separate fibres are thrown into sudden and forcible contraction, and the balance of the muscle is destroyed. If the irritation is slight it may be excited by tapping the muscle or exposing it to cold.

CONTRACTURE. If the irritative condition is continued for a long time, or the inhibitory control of the brain over the cord is suspended for a similar period, the limb will assume a fixed position due to one of three possible conditions. The paralysis of one group of muscles will allow the unopposed over-action of the antagonists, or the irritation of one set of muscles will overcome the normal tonicity of their opponents, or long-continued, abnormal tension of a set of muscles will induce a fibro-tendinous change in the structure of the over-strained muscles, which will be followed by contraction. In the first two instances the rigidity will cease during sleep, and the position may be overcome by continued force, when the contracture will suddenly give way. This is called "lead-pipe" rigidity. In the latter case pressure will reduce the contracture to some extent when the opposition to further reduction will prove irresistible.

SKIN-REFLEXES. These are not as well understood as are the

tendon-reflexes, and their centers may be in the cord, but have not been localized. They are often absent, and are not always capable of interpretation. They are usually lost when the tendon-reflexes are exaggerated, and in disease of the brain the absence on one half the body, and their presence upon the other, points out that the side without the reflex is opposite to the lesion in the brain. Skin reflexes are never lost in hysteria, thus again differentiating it from organic disease, but are exaggerated if the centers are irritated or inflamed, and in many functional conditions marked by diminished nervous tension they may be inhibited to a degree by volition.

BABINSKI'S REFLEX. The plantar reflex is exaggerated in many conditions, and is one of flexion, but if the cause of exaggeration is an organic one stroking the sole of the foot (perhaps the stimulus will need to be a somewhat painful one) the reflex will be an over-extension of the great toe, and perhaps of all the toes. Under the same conditions the like response will result from the stroking in a downward direction of the skin on the inside of the calf. This is called Oppenheim's reflex.

SENSORY DISTURBANCES. Just as the irritation of motor tracts produces an increase of function, and a destruction of them a paralysis (and of cells the same), so we find in the sensory sphere hyperesthesias and paresthesias from irritation, and anesthesias from destruction. These are sensory illusions, but the irritant is mentally localized in the skin-areas to which the injured nerves were distributed, and whence the stimuli would usually come.

ATAXIA. We retain our ability to balance ourselves in an upright position, and to perform movements with accuracy, and to correctly appreciate the position of our limbs, in absence of information from vision, by means of the sensations conveyed to the central organs. These sensations from the skin are tactile impressions, while those from the joints, muscles and tendons are those of pressure and tension. If this information is deficient the person will present some grade of ataxia and incoördination, and he will also have some grade of asteriognosis. This ataxia or incoördination will be the result of any lesion of the cells or tracts relating to the appreciation or transmission of common sensation, but will be most pronounced when the fibres or cells concerned in the function of muscle and joint-sense are injured. Locally considered we shall find ataxia if there is a lesion of the parietal lobe, and it often has been found when the motor cortex has been the seat of a lesion, since over much of its area there is an admixture of sensory cells. It will follow a lesion of the posterior part of the internal capsule, and has followed a lesion of the corpora quadrigemina. A lesion in the crus which is capable of producing an oculo-motor palsy with hemianopsia will also produce some degree of ataxia. A lesion in the pons which produces anesthesia in the

distribution of the fifth nerve or a facial palsy will induce ataxia. A lesion in the medulla which causes deafness or a hypoglossal palsy will also produce an ataxic condition.

ATROPHY. The nutrition of the various parts of the body is regulated by cells lying in the anterior horn of the cord. Some have deduced from post-mortem findings that this is a specialized function of a definite group of cells lying inside and nearer to the central canal than those which are purely motor in function, while others consider that nutrition is merely an accessory function of the motor cells in whatever portion of the anterior horn they may happen to lie. In any case, it is certain that any structure that is cut off from the influence of its segment of the anterior horn-cells will atrophy. If this cell-destruction is a gradual process the structures will gradually waste, but if sudden, as in inflammatory conditions, the atrophic process will be so intense that sloughing will ensue as is seen in myelitis, hemorrhage into the cord, and less frequently, after intense shocks and degenerative processes in the brain.

THE SYMPATHETIC NERVOUS SYSTEM.

ANATOMY. The sympathetic nervous system is not an independent structure, but is a somewhat specialized portion of the central nervous system. It may be divided into three divisions, viz., a chain of from twenty to twenty-three inter-connected ganglia lying upon the sides of the vertebral column; the pre-vertebral plexuses, cardiac lying above the heart and supplying it, solar lying behind the stomach and supplying it, and the abdominal viscera and their vessels, and the hypogastric which lies between the two iliac arteries, and supplies the pelvic viscera; secondary, and isolated plexuses, often of minute size, which are in or near various organs, and preside over their functions, e.g., in the heart, uterus, supra-renal bodies and intestines. Each of these ganglia is reddish gray in color, and soft in consistency, but is enclosed in a tough fibrous capsule. A ganglion is made up of nerve-cells, and medullated, and non-medullated fibres. The ganglia are connected with the spinal cells and spinal nerves by tracts which are called rami communicantes, with one another by rami internodiales, and with the subsidiary plexuses by rami peripherales. The rami communicantes are found only in connection with the thoracic, and first (and sometimes the second) lumbar nerves, the second, third and fourth sacral nerves, and possibly the third, seventh, ninth, tenth and eleventh cranial nerves. These rami communicantes are the avenues of communication between the ganglia and the spinal cord, and contain medullated fibres which are derived from the motor and sensory

cells of the cord, and arborize about the cells of the connected ganglion, or one in the vicinity, in which latter contingency they pass through the first ganglion on their way to the second. They also contain non-medullated fibres which arise from the cells of the sympathetic ganglion and pass to the cord, to preside over its vegetative functions, or pass thence to their destination in the spinal nerves. The rami internodiales are simply commissures between contiguous ganglia. We find then that the appearance of the ganglia near the spine is that of oval bodies connected to the spinal cord, and also with the ganglia above and below them and sending off a separate branch of communication with the less closely related ganglia. The non-medullated fibres are, in place of myelin, covered by a thin fibrous envelope with cells at intervals lying between the envelope and the axis-cylinder. These are called the fibres of Remak. The connection of the sympathetic nervous system with the brain is by means of the pneumogastric nerve.

PHYSIOLOGY. The sympathetic system is not a separate reflex center, but is so closely related structurally to the brain and cord that we must believe that its functional powers are derived from these higher centers, and are modified in quality and direction only in their passage through the ganglia. Under certain circumstances they are evidently capable of complicated reflex action, since parturition has been successfully accomplished by the action of the lumbar centers when the cord has entirely been severed above that point. Their inter-relation is shown by the fact that all the vegetative functions are seriously disturbed by conditions which entail severe shock to the brain or cord, and conversely it is as true that a perversion of the vegetative processes may disturb or totally inhibit the activity of the brain and cord. Whether this result is a direct one, or whether it is the secondary result of intoxications arising from the interference with the vegetative functions is not yet known. They are, however their powers are originated, the centers of motor, vaso-motor, secretory and (perhaps) trophic impulses. They preside over the vegetative functions of life through their action upon unstriped muscle-fibre. As an exception to this rule it may be stated that they innervate the striped muscle of the heart. Hence the viscera are under their control, the dilators of the pupil, the unstriped fibres of the lids, the erectores pilorum, and the sweat and salivary glands. They also possess inhibitory functions over various structures and secretions.

PATHOLOGY. The pathology of this system is as yet in doubt, and also the diseases resulting from lesions of these nerves. Most of the conditions supposed to flow from such a cause seem possibly to be produced from disturbance of its vaso-motor function alone, and in other cases, like glaucoma and goitre, where changes have

been found in the sympathetic system the lesion has not been a constant one, and a lesion in the central nervous system has at times been competent to produce an exactly similar condition.

ETIOLOGY OF NERVOUS DISEASES.

The causes of nervous disease are injuries, exposure, poisons, toxemias, infections, and exhaustions of tissue from over-use. A neuropathic heredity is the basis of a large proportion of nervous diseases, and it may manifest itself in a deficient development of the tissues, whether they be the cells, tracts, or connective tissue elements, and these deficient structures are the site of later degeneration.

PATHOLOGY OF NERVOUS DISEASES.

The forms of disease affecting the nervous system are: (1) Malformations; incomplete development, or agenesis; defective development, or dysgenesis; these are due to disease "in utero"; (2) Hyperemia, anemia, hemorrhage, edema, and arterial and venous disease; (3) Inflammations; (4) Degenerations and atrophy, softening, sclerosis, and gliosis; (5) Infections like tuberculosis and syphilis; (6) Tumors and parasites; (7) Nutritive and functional disorders, including disorders associated with metabolic and glandular defect, such as acromegaly, and exophthalmic goitre. Since the nervous system is composed of nerve-cells and fibres, connective tissue, neuroglia, blood-vessels and lymphatics, and the lymph-spaces of His which are peculiar to nervous tissue, any disease process will be manifested in a structural or functional change of one or several of these tissues, for no process strictly confines itself to one of them, and generally involves them all. The symptoms of disease of the brain are similar in character, in spite of differences in the pathology of the process, since they depend far more upon the localization than upon the kind of disease-process primarily causing the injury of the nerve-elements. The actual changes to which these elements are subject are few, but different localizations and combinations of lesions give rise to numerous well-marked symptom-complexes. Since the blood circulates more abundantly, and at a more rapid rate in the anterior and central parts of the cord than in the posterior area, we should expect the former areas to be more resistant to disease, and this is clinically the fact. In diseases of the cord, sensory degenerations are always systematized, but if motor they are liable to be more diffuse. The brain is often affected by functional diseases (practically always nutritional or toxic), while the cord is practically immune to such conditions, except in company with the brain.

Both brain and cord are equally subjects of inflammation and injury, but the cord and its meninges are more resistant to infections than is the brain and its envelopes. As a general truth it may be stated that brain conditions are almost always vascular in origin. Intoxicants sometimes damage the cells of the cortex and the nuclei, so that there is a primary degeneration of the neuraxon, but much more often this is the secondary result of a primary vascular change. The changes of mentality and function incident to old age are generally arterio-sclerotic, but may be due to a primary disintegration of the cells from the inability of these structures to longer continue the processes of metabolism to the degree necessary to healthy function. Thrombosis is more common, and leads to more serious results in the brain. The course of the vessels in the brain favors both thrombosis and embolism. The brain is often affected by abscess, hemorrhage, and tumor, while the cord is rarely subject to such damage. When the causative condition is a developmental fault, the process which results from some exciting cause is a productive inflammation, and the imperfect structures indulge in excessive cell-proliferation, which results in a pressure-necrosis of the true nervous tissue in the vicinity. The final result is a mass of sclerosis cutting off tracts, and replacing cell-areas, or softening and absorption of the new tissue ensues and a cavity is produced. This is the pathology of syringomyelia, which is a gliomatous overgrowth from the walls of the central canal of the cord. Multiple sclerosis is thought by some to be a developmental fault in the myelin sheath of the nerve-fibres. After their degeneration and absorption, there is a replacement growth of neuroglia, and spots of sclerosis result which are scattered in a random manner throughout the brain and cord. Generally in this disease we find the community of structural change referred to in the introduction, since blood-vessels often show occlusion, neuroglia an irritative overgrowth, and the nerve-fibres are degenerated. Hyperemia and anemia are the cause of functional changes which may be severe and widespread in their effects. The nerve-cell is absolutely dependent upon the constant integrity and perfection of circulation, while the fibre is resistant to changes of the same degree for a long time, but cannot endure dissociation from its nutrient cell for an instant. This is probably the primary cause of degenerations in the brain and cord, although the gliomatous overgrowth clouds the post-mortem appearances. This may be simultaneous, and dependent upon the same nutritional fault. Toxins and poisons may set up the same changes, and are also nutritional, although the fault in such a case is not the volume, but the character of the blood. Edema and vascular disease destroy nervous tissue by infiltration and pressure, or by producing an ischemia, which latter produces the changes referred to

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above. Inflammation in any structure of the body is the tissue response to a tissue irritant, and it may be due to a chemical or toxic irritant, or to pressure. When the irritant is removed it tends to subside, but since it affects not only blood-vessels and nervous tissue, but interstitial structures also, the result may be a proliferation of tissue which destroys nervous tissue which is not capable of repair, and permanent damage may be the result. The primary attack is a thickening of the coats of the arteries, and a subsequent infiltration of the surrounding tissues by an invasion of round cells. Inflammations are both exudative and

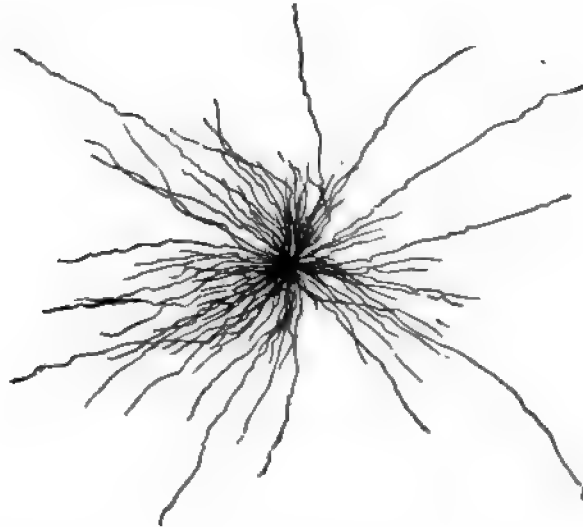


FIGURE 8.—GLIA CELL.

From the spinal cord of a child nine months of age. Highly magnified. (Lenhossek.)

productive. An exudative inflammation may be so slight that it does not produce any necrosis, or it may destroy large areas in this way. It may be purulent, and still leave the tissues capable of repair, but usually necrosis follows closely on its appearance. Most exudative inflammations are acute or subacute, and the usual forms of edema are exudative inflammations. In primary meningitis there is every evidence that the initial change is a change in the arteries, and an intense congestion. Since the periphery of the brain and the cord is nourished by short terminal arteries, there is a softening of the cortex of the brain, and the periphery of the cord, for a depth of half a centimetre. Myelitis is an instance of a similar condition in the substance of the cord. Here the vessels are congested, and swollen, and degenerated, they

finally rupture, and leucocytes and round cells swarm throughout the tissues, and infiltrate the cells, and we find not only a primary degeneration of the nerve-fibres, but also after the myelin sheaths have been infiltrated they degenerate and expose the nerve-fibres to compression by the exudate, and they break down in secondary degeneration. Productive inflammation has a chronic course with little congestion or exudation, and new connective tissue is formed from the first. Tuberculous and syphilitic processes are instances of it, and it is also caused by alcohol, lead, arsenic, and other metallic poisons; by toxins and auto-toxins, seen in gout, rheumatism, diabetes, and states of inanition. The primary change affects the blood-vessels, lymphatics, or connective tissue, and some have considered that all such changes are the results of a primary overgrowth of neuroglia, a gliosis.

Degeneration means the destruction of the parenchyma of an organ, and it may be acute or chronic, primary or secondary. If it be acute, we find softening or necrosis, due to the cutting off of the vascular supply, to direct injury, to necrosis, or to poisons causing inflammation. It may be followed by repair. Chronic degeneration is accompanied and followed by a connective-tissue overgrowth, and the production of a sclerotic area. It is a replacement process, and may go hand in hand with degeneration. If degeneration is primary, it is nutritional, or from a toxic poison which affects the fibre or cell directly. Secondary degeneration is that in which a fibre or cell is cut off from its center of nutrition, or there is an interruption of its vascular supply, but generally we refer to cases where the fibre has been cut off from its cell. Primary and secondary often occur in the same disease. Degenerations result from arsenic, lead, phosphorus, and other metallic poisons, the toxins and infections, from obliterative arteritis which occurs in old age, or from an inherent defect in cell-nutrition. The primary form is seen in the cord in progressive muscular atrophy, myelomalacia, tabes dorsalis, and other system diseases, while the secondary is seen in the lateral sclerosis which follows a hemiplegia, chronic myelitis, or syringomyelia. In the brain the primary degenerations are due to such changes as reduce the blood supply to the cells (like arterio-sclerosis and arteritis), and to the effects of toxins which have a selective affinity for nerve-cells, while the secondary degenerations result from such lesions as will cut off the fibres in their course from one set of cells to arborize about another, either in another part of the brain, or in their course to the cord. The selective action of poisons and toxins should be noted. For no assignable reason, particular agents affect one set of structures, and leave the others unaffected, and clinical experience is the only basis for deducing the probable cause of many intoxications. The peripheral nerves are subject to interstitial inflammations, like connective tissues in any other

part of the body; they are peculiarly liable to secondary degeneration from infiltration of the fibres by toxic exudates from the membranes of the meninges of the brain and the cord, since these structures form their envelopes as they pass out from the skull and the spinal canal, and the lymph spaces of the nerves are continuous with these perineural spaces thus formed. The fibres are exposed to primary degeneration from the specific action of toxins and intoxicants circulating in the blood, which selectively act upon nervous structures at their weakest point, i.e., at the periphery, which is the portion farthest removed from the nutritive cell. Refrigeration is also very potent at this point. They are also especially liable to secondary degeneration from mechanical separation by injury. Hemorrhage acts by direct destruction of the nervous elements, and by infiltrating surrounding parts, some of such areas always being the site of necrosis, and by injurious pressure upon a still larger area, and functionally disturbs the whole brain by the sudden change of blood-pressure, producing molecular shock to the cells of the brain. The effect is greatest when the disturbance is a sudden one, the element of quantity being of secondary importance. A nerve-cell once destroyed is never regenerated, and, while nerve-fibres sometimes persist through a sclerotic patch, we have no data upon which to base an opinion that such a patch is ever absorbed, unless it be syphilitic in origin. Peripheral nerves, however, may regenerate, even after a long interval. Degeneration in a severed nerve-fibre begins in about twenty-four hours, and extends about a quarter of an inch toward the nutrient cell, while the whole peripheral portion is wasted. Union is probably never possible by coaptation of the severed portions, but is always by regeneration. Through the intervening connective tissue new nerve-fibrils reach out toward the periphery, and, if the interval is not too great, they will reunite with the peripheral portions of the fibre, the myelin is re-formed, and restitution of tissues, and restoration of function ensue.

TRACTS OF THE CORD.

The sensory nerve gains its impressions of the external world by means of the superficial inter-epithelial arborizations and end-organs just described. From some one of these collectors of external impressions the sensory nerve courses (centripetally as it is called) in the trunk of a mixed nerve, until it reaches the ganglion which lies on the pedicle of the vertebra. This posterior root-ganglion is made up of cells which are bipolar, and each one of which nourishes one nerve-fibre from each end, the one extending from the surface to the ganglion, and the other from the

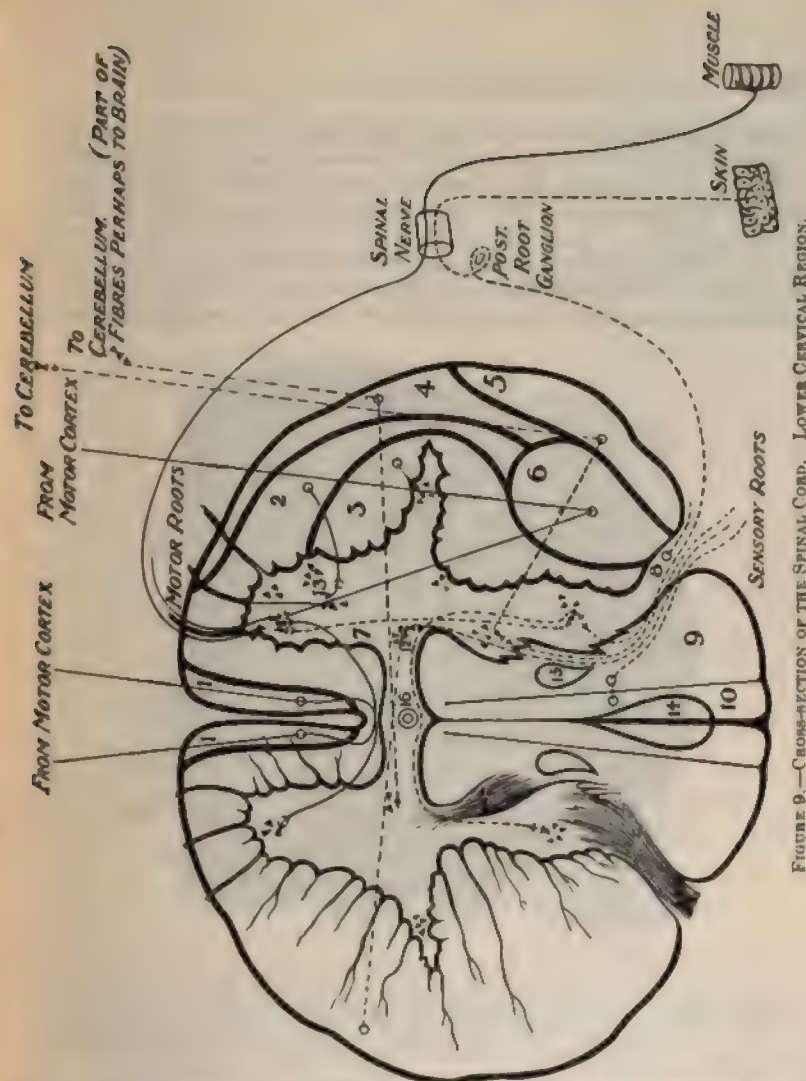


FIGURE 9.—CROSS-SECTION OF THE SPINAL CORD, LOWER CERVICAL REGION.

1. Direct Pyramidal tract (inhibitory (?) to motor cells *opposite side*). 2. Antero-lateral Ground-bundle (association tract, connecting contiguous levels of the cord). 3. Lateral Ground-bundle (association). 4. Gowers' tract (conveying impressions of pain and temperature, derived from the cells upon the *opposite* side of the cord, about the central canal (12), upward to the cerebellum and, possibly, to the brain). 5. Direct Cerebellar tract (conveying to the cerebellum impressions of pressure and tension on joints, muscles and tendons, derived from the cells of Clarke's column, unmarked, but seen at the inner aspect of the base of the posterior horns. These cells are on the *same* side of the cord as the column). 6. Crossed Pyramidal tract (conveying downward from the brain voluntary impulses to motion to the cells of the anterior horn of the *same* side). 7. Anterior horn (seat of motor and trophic cells). 8. Posterior nerve-roots (entrance of sensory fibres to the cord. This also covers the site of Lissauer's association tract which diffuses all sensory impressions through several adjoining segments). 9. Column of Burdach (the site of the first distribution of sensory fibres and of the change of some to a vertical direction). 10. Column of Gull (formed of fibres derived from the column of Burdach which are passing upward to the medulla and brain). 12. Cells about the central canal (recipients of sensations of pain and temperature). 13. Motor, and perhaps also trophic, cells in the anterior horn. 14. Septo-marginal tract. 15. Comma tract of Schultz. These two are descending tracts which do not degenerate with the rest of the posterior columns. 16. Central canal. When a circle is shown in a column it denotes that the fibre there changes its direction from horizontal to vertical, or vice versa.

ganglion to the cord. That fibre which goes to the cord enters it quite directly by the column of Lissauer (this is a commissural column, and as such is typical), or goes to form a narrow band (*bandelette externe*) in the column of Burdach, just central to the posterior horn. Many of the columns of the cord go directly up or down throughout its whole extent, and the fibre at the bottom may be identified with the same fibre at the top, or vice versa, but there are other columns whose fibres run but a short distance, and

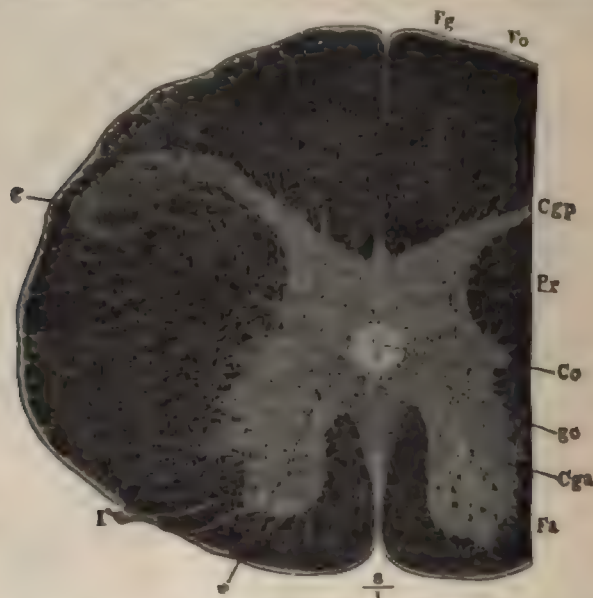


FIGURE 10.—SECTION OF THE CORD AT THE LEVEL OF THE FIRST CERVICAL NERVE.

The ascending posterior columns, *Fg* and *Fc*, have increased greatly. The posterior columns are clubbed at the ends and show masses of gray matter (the gray substance of Roland). *Fg* and *Fc* are just about to terminate respectively in the nucleus gracilis and cuneatus on the posterior aspect of the medulla. *Cgp* is the posterior horn and *Cga* the anterior. *Cc* is the central canal and *gc* the glia substance about it. (From Henle.)

then go horizontally to end about some cell in a segment, or several segments, removed from the site of its original appearance in the tract. Such fibres are commissures, as they tie together different segments, and different levels of the cord, for some associated purpose. The general vertical extent of a fibre in the column of Lissauer is three to four segments. From the *bandelette* the sensory fibres go in four directions:

1. Into the column of Burdach, in which they ascend, but tend always to go to the region of the median fissure, and therefore by the time they have ascended to the extent of a few segments they

are nearing the central fissure, and constitute the column of Goll. New axons (sensory nerve-fibres) are all the time entering the cord, so that the column of Burdach is gaining from the periphery, while it is losing toward the center. These fibres convey common sensation.

2. Other fibres tend externally, and arborize about the cells in the posterior horn of the side of the entrance, some by the posterior commissure to the other posterior horn, where they



FIGURE 11.—SECTION AT THE LOWER PART OF THE MEDULLA.

This is at the upper margin of the motor decussation. The fibres from the lateral tracts are seen crossing over to join the uncrossed pyramidal tracts to form the Anterior Pyramids of the Medulla. Thus decussation cuts off the anterior horns. At *Ng* in the column of Goll (at the top of the figure) is seen the beginning of the formation of the nucleus gracilis; *g* is the substance of Roland or gelatinosa. *XI* is the root of the eleventh nerve. (From Henle.)

arborize about the cells found on that side. From the cells of the posterior horn, about which some of the fibres have been said to arborize on the same side as that on which they enter, new axons are sent off, which in their turn arborize about the cells (motor) in the anterior horn of the same side, and thus form the sensory side of the reflex arc. All these fibres convey common sensation. Below are the principal reflexes, although the number of actual ones is only equaled by the number of muscles. The spinal center for the plantar reflex is located in the 2d and 3d

sacral. Ankle-jerk and clonus are in the 1st sacral and 5th lumbar. Knee-jerk is in the 2d and 3d lumbar. Elbow-jerk is in the 5th, 6th, and 7th cervical.

3. Other fibres go up and arborize about the cells of the vesicular column of Clarke, situated on the internal aspect of the base of the posterior horns. This column of cells extends from the 7th cervical to the 3d lumbar, and has an analogue in a similar column of cells in the sacral cord, called the nucleus of Stilling. From these cells new axons are sent out, which stream across the gray matter of the posterior horns, until they gain the extreme posterolateral margin of the cord, where they take a vertical direction, and go up to the cerebellum under the name of the direct cerebellar tract, which extends from the 8th or 9th thoracic to the cerebellum. This is on the same side as the cells from which the axons are derived. Conveys joint, tendon, and muscle sensation.

4. Still other fibres arborize about a collection of cells which is found upon each side of the central canal of the cord. These cells send out new axons, which cross in the anterior (white) commissure, and assume a vertical position on reaching a position in a tract in the antero-lateral portion of the cord. The tract of Gowers lies on the antero-lateral margin, while another, so-called antero-lateral tract, is situated internal to it, and extends more anteriorly. Some have decided that the tract of Gowers after extending upward to the pons, then curves over and enters the cerebellum in the peduncle. Believing this, they cannot grant that the fibres conveying impressions of pain and temperature can ascend in this tract, since lesions of the parietal cortex have been known to cause this loss. Mott believes that while true of part of the tract, it is not true of the whole, but that a part of the fibres end in the optic thalamus, which is in communication with the parietal cortex. This tract extends the full length of the cord.

The fibres are thus grouped in the cord in tracts, each one of which has a certain definite function, and when disease shows a selective affinity for one or another of these, to the exclusion of the others, we speak of the condition as a "System Disease." They are then during their passage through the cord "systematized," i.e., grouped in a definite bundle, but after reaching the medulla are re-arranged. In the medulla the fibres of the column of Goll arborize about that collection of cells known as the nucleus gracilis, while the fibres of the column of Burdach end about the cells of the nucleus cuneatus. These nuclei lie on the posterior aspect of the medulla in the same relative position as the two columns hold in the cord, i.e., gracilis toward the center, and cuneatus outside of it.

In the top of the direct cerebellar tract is also developed a nucleus which is called the inferior olivary body. (The superior olivary body lies in the medulla at the level of the facial nucleus,

and is probably the center for the re-arrangement of the fibres from the cochlea).

The sensory decussation occurs in that structure known as the *formatio reticularis*. This structure extends from the medulla through the pons, and into the crus, where it dies out, as a structure, since the fibres concerned in its make-up are now composing, with others, the internal capsule. The *formatio* is an area made

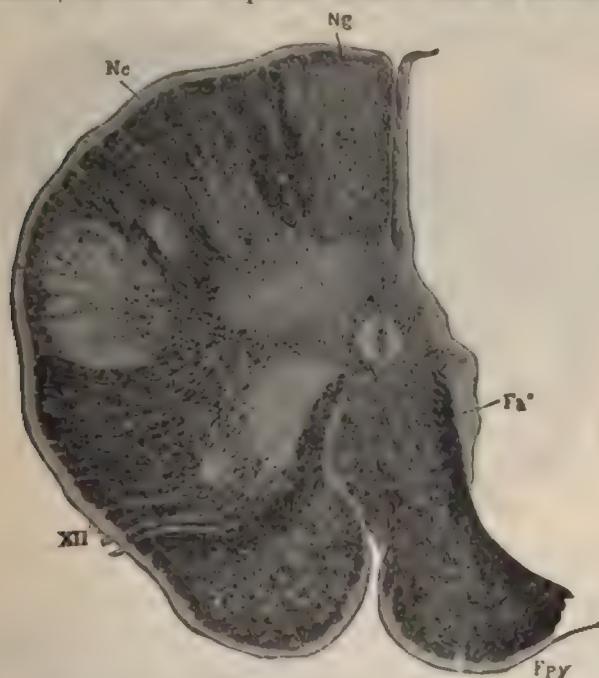


FIGURE 12.—SECTION OF THE MEDULLA AT A LITTLE HIGHER LEVEL THAN THE PRECEDING.

The decussation is just beginning (if we think of the motor fibres as descending, as is the fact). The nucleus gracilis, *Ng*, occupies nearly the whole space formerly occupied by the column and the nucleus cuneatus (from column of Burdach) also largely replaces that column. *XII* is the twelfth nerve arising from a nucleus which is located at the site of an anterior horn at a lower level. (From Hende.)

up of three sets of fibres and cell-groups interspersed among them. The fibres found are:

1. Commissural fibres of the medulla, pons, and cerebellum.
2. Cranial nerve-fibres which have a vertical direction.
3. Sensory-fibres which have a longitudinal direction. In the meshes between these fibres lie isolated cells, and also groups of them. These groups are:

1. Nucleus ambiguus, of the 9th and 10th cranial nerves.
2. Superior olive, from which arise the 6th and 8th.

3. Nuclei of the lemniscus, a sensory group tending toward the brain.

4. Red nucleus. This lies near the aqueduct in the region of the crus, and is a nucleus into which come fibres from the anterior corpus quadrigeminus of the opposite side; a few fibres from Gudden's commissure; fibres from the optic thalamus, and from the lenticular nucleus, the latter fibres coming by way of the

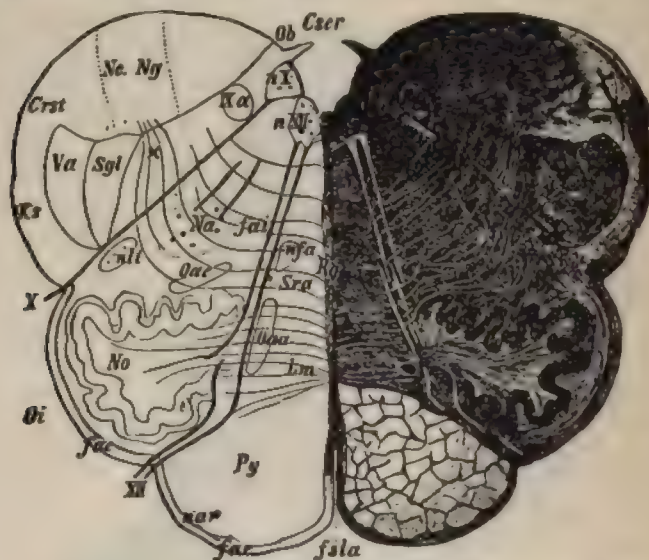


FIGURE 13.—SECTION OF THE MEDULLA THROUGH THE CALAMUS SCRIPTORIUS.

The Pyramids, *Py*, have been completely formed. *No* is the inferior Olive; *Im* is the Lemniscus; *Va* is the ascending root of the fifth nerve; *Ka* is the Direct Cerebellar tract; *Crst* is the Restiform body which becomes the Inferior Peduncle of the Cerebellum. *Na* and *NaXI* are the nuclei of the tenth and twelfth nerves which nerves are seen to emerge at the margin of the figure. *IXa* (just posterior to these nuclei) is the ascending root of the ninth nerve. Tracing the fibres downward from *X* at the upper portion, we may see how the sensory decussation, or crossing, is accomplished at this point. The opening under *Cser* (Calamus Scriptorius) is the beginning of the Fourth Ventricle. (From Obersteiner.)

internal capsule. On the lower side, fibres enter it by way of the superior cerebellar peduncle from the cerebellar hemisphere of the opposite side.

This formatio reticularis contains a long ascending tract, as some fibres pass up through it on their way to the brain, while others come to a certain level, and then arborize about some of the cells, while still others originate in these same cells, and go thence to the brain. The tract of Gowers certainly comes up into the formatio, but its subsequent course is not certain, though it is probable that it curves over the 5th nerve, and enters

the cerebellum by way of the superior cerebellar peduncle, and the velum medullare anticum, superior medullary velum, and valve of Vieussens, which is an arch of white matter binding together the lower surfaces of the two superior cerebellar peduncles. Another part of these fibres of the tract of Gowers goes upward from the formatio, and terminates in part in the corpus quadrigeminus, and part in the optic thalamus, the cortical representation of both groups being in the parietal lobe, the fibres passing up with those of the mesial fillet. That part of the tract of Gowers which we traced into the cerebellum arborizes there about the cells of

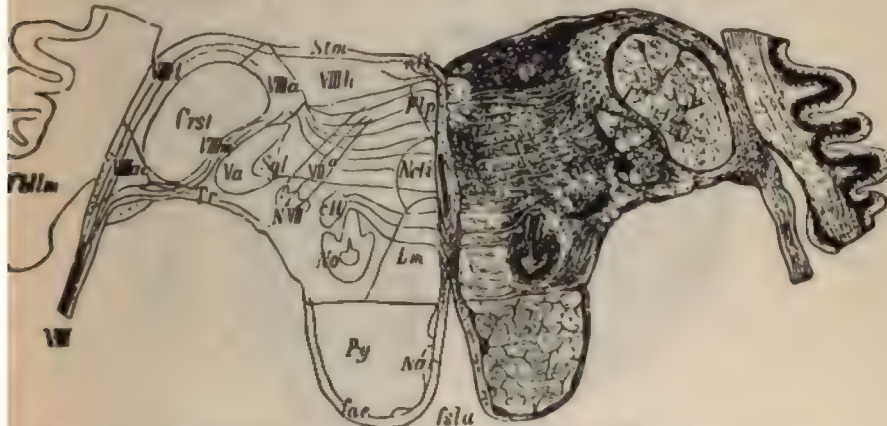


FIGURE 14.—SECTION THROUGH THE MEDULLA AT THE SITE OF THE STRIAE ACOUSTICAE (SUPERIOR OLIVE).

The floor of the fourth ventricle is widened out here and the overlying Cerebellum has been cut away, except at the sides, where it appears, *Chm*. The Restiform body is surrounded by the roots of the eighth nerve which arises from *VIII h*, its chief nucleus. *VIII l* is its lateral nucleus; *VIII m* its median nucleus; *VIII ac*, its accessory nucleus. *N VII* is the nucleus of the seventh nerve; *VII a* fibres ascending from the same; the other signs indicate parts mentioned before. (From Obersteiner.)

Purkinje, from which cells axons descend in the inferior cerebellar peduncle, and penetrate the inferior olive by way of the restiform body, and thence send down fibres in the tract of Marchi and Lowenthal, from which the fibres cross in the anterior commissure of the cord, and arborize about the motor cells in the anterior horn.

Ascending sensory fibres, generally speaking, gain the sensory cortex either by way of the thalamus, or more directly by way of the internal capsule.

In the outer third of the formatio is a collection of peculiar cells, in which end the fibres of the descending or sensory tract of the 5th nerve. These cells are situated in substantia Rolandi, which is the upward extension of this peculiar substance, and the

collection of cells which we have found under the same name in the posterior horns throughout their whole extent. These fibres enter the pons at about its middle, and then bend downward. The outer side of the formatio is then the seat of the cell nuclei having to do with sensation for that same side in the face and head.

The main portion of the fibres arising from the nuclei gracilis and cuneatus goes in this sensory decussation, formatio reticularis, from the back of the medulla toward its front, to a position just

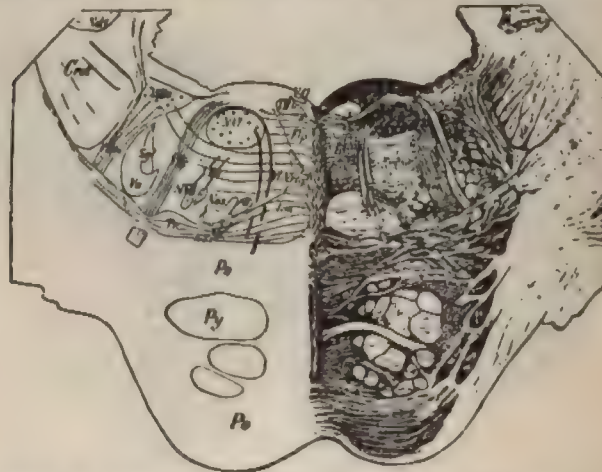


FIGURE 15.—SECTION OF THE PONS WITH THE CEREBELLUM CUT AWAY.

The separation of the fibres of the Pyramids, on account of the interposition of the interlacing fibres of the middle peduncles of the Cerebellum, is shown. The upper part of the nucleus of the seventh appears at VII. VIIb shows a cross-section of the ascending fibres of the same nerve. NVI is the nucleus of the sixth nerve, the knee being at a higher point. Ndt is the Dentate nucleus of the Cerebellum, and Flp is the Posterior Longitudinal Fasciculus. Crat is the former Restiform body which is now the Inferior Peduncle of the Cerebellum. Lingula. (From Obersteiner.)

back of the pyramid of that side. This bundle of sensory fibres during this process is called the internal arcuate bundle of fibres. On reaching its new position it at once assumes a vertical direction, and proceeds toward the brain, and is now called the mesial fillet or lemniscus. It now lies just back of the pyramids (the descending motor tracts) in the front part of the medulla. In the pons it sinks a little toward the rear, but by the time it has passed the limits of the pons in its upward course, and has come to the mid-brain, or region of the crus, it trends backward, and the two halves lie each side of the iter, and, at the lower margin of the corpora quadrigemina, each lemniscus (here called the mesial fillet) divides into two parts. The major part goes to the parietal and post-

central cortex by way of the optic thalamus, and the other to the same ultimate destination, but more directly, by way of the internal capsule, occupying the posterior third of the posterior limb. (Goes to the optic thalamus by way of the post. corpus quadrigeminus.)

We have traced the major portion of the fibres arising from the nucleus gracilis and cuneatus to their final distribution on the parietal cortex, but we must also remember that in the structure

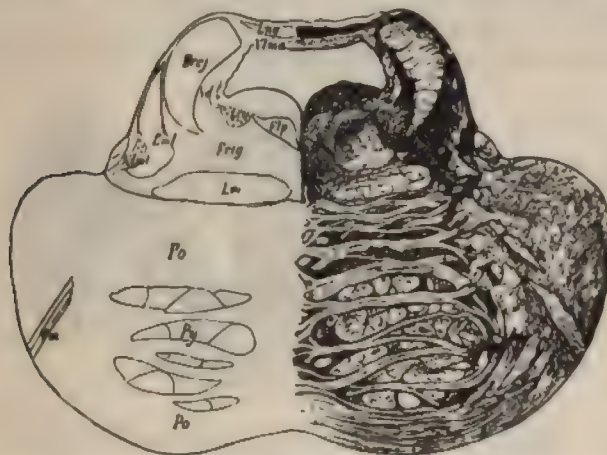


FIGURE 16.—SECTION ABOVE THE CEREBELLAR STRUCTURES.

The Pyramids are still in separated bundles amid the decussating fibres of the middle Peduncles of the Cerebellum. *Lm* is the median Lemniscus; *Plp* is the Formatio reticularis. *Plp* is the posterior Longitudinal Bundle. *Breg* is the superior Peduncle of the Cerebellum. *Vlma* is the Velum medullare anticum, and *Lmg* the Lingula. *Vm* is the outgoing root of the fifth nerve. The Lemniscus (sensory bundle) has now been separated into two sections, the lateral being composed of auditory fibres, and going to the region of the Corpora Quadrigemina, and the median (common sensation) going to the parietal cortex. (From Obersteiner.)

of the medulla, where they decussate, they are also known as the internal arcuate fibres.

Two other smaller bundles leave these nuclei. The first passes around the *posterior* periphery of the medulla, and dips into the olive of the *same* side, which is equivalent to saying that it joins the restiform body of the same side, which is prolonged into the cerebellum as the inferior cerebellar peduncle. This bundle is one of the two groups known as posterior external arcuate fibres.

The second group from the same source, after crossing in the raphe, formatio reticularis, passes forward about the periphery of the pyramids, and enters the olive of the *opposite* side, and so joins the restiform body of that side. These are known as the anterior external arcuate fibres.

Summary of the arcuate fibres:

1. Internal. The majority of the fibres from the nucleus gracilis and cuneatus, which cross directly to a position back of the pyramids of the opposite side.

2. Posterior external. This goes about the posterior periphery of the medulla to the olive of the *same* side.

3. Anterior external. Cross in the raphe, and then go about the external anterior surface of the pyramids, and enter the olive of the *opposite* side.

The direct cerebellar tract, as has been said, ends in the nucleus called the inferior olive, and from the cells of this nucleus new

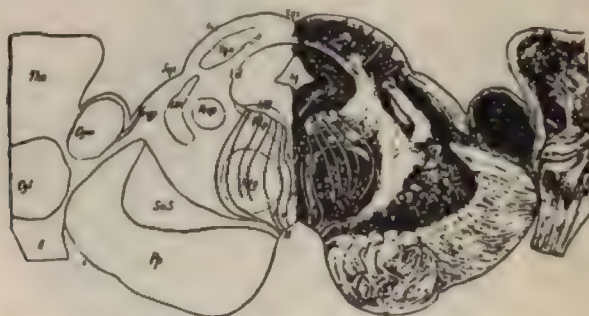


FIGURE 17.—SECTION THROUGH THE ANTERIOR PAIR OF THE CORPORA QUADRIGEMINA, THE CRURA CEREBRI, THROUGH PART OF THE OPTIC THALAMI, AND OF THE EXTERNAL GENICULATE BODIES.

Py is the Pyramid, which occupies the middle only of the lower part, or Crus. *Cgt* is the internal Geniculate body; *Cgt* the external; *Tha* is the Optic Thalamus; *Il* is the Optic tract; *Sns*, Substantia nigra; *Nty*, the Red Nucleus of the Tegmentum; *III*, fibres of the third nerve (Oculo-motor); *N III*, Nucleus of the third nerve; *Lm*, Median Lemniscus; *Ag*, Aqueduct of Sylvius. About the Aqueduct is a mass of gray matter at whose outer border is the descending root of the fifth nerve. Below the Aqueduct and lateral from it, is the nucleus of the third nerve, whose fibres are seen emerging just between the Crura. The lateral Lemniscus has disappeared, having entered the Corpora Quadrigemina. The large mass of the Substantia nigra separates the Crus from the parts above (all above being termed the Tegmentum). (From Obersteiner.)

axons extend in the restiform bodies in the inferior cerebellar peduncles, and end in the superior vermis or worm of the cerebellum. It is well in passing, to note that the inferior cerebellar peduncle contains also the two bundles of fibres from the nucleus gracilis and cuneatus which we have just described under the name of the posterior external and anterior external arcuate fibres; also small bundles of fibres from the lateral nuclei, which are composed of cell groups from the anterior horns with some local enlargement; also the descending cerebellar tracts which are axons of the cells of Purkinje, and form the other part of the reflex arc with the columns of Gowers (these are the columns of Marchi and Lowenthal); also the direct cerebellar sensory tract from Deiter's nucleus in the medulla, where are gathered impressions

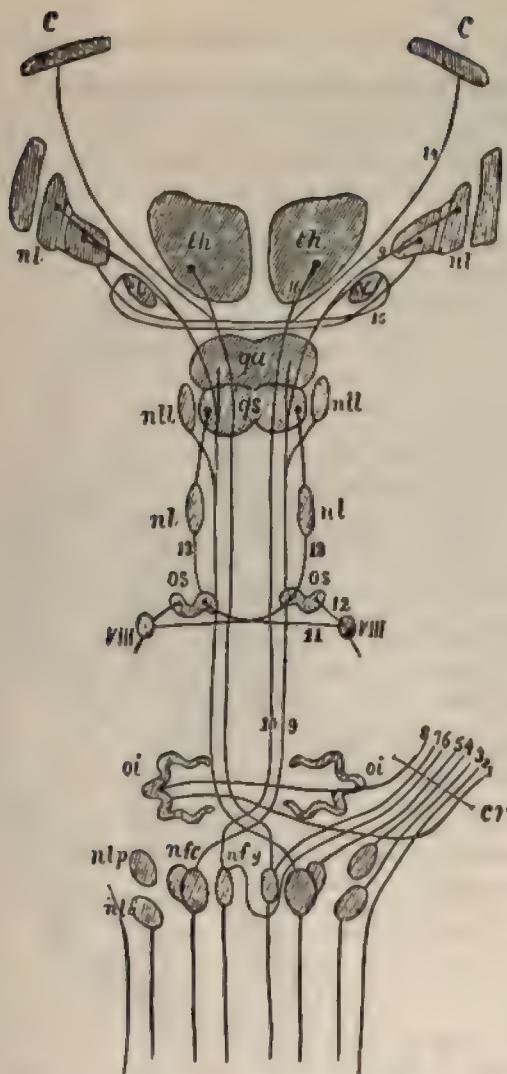


FIGURE 18.—A SCHEMATIC REPRESENTATION OF THE COURSE OF FIBERS FROM THE NUCLEI OF THE POSTERIOR COLUMNS OF THE SPINAL CORD, TO AND THROUGH BOTH LEMNISC, AND TO THE CEREBELLUM.

C, the cortex; *nl*, the lenticular nucleus; *th*, the optic thalamus; *cl*, Luy's body or corpus subthalamicum; *ga* and *qs*, the corpora quadrigemina; *nl*, nucleus in the lateral lemniscus; *os*, the superior olive; *VIII*, anterior acoustic nerve nucleus; *oi*, the olivary body; *nla*, nucleus of antero-lateral column; *nlp*, nucleus of postero-lateral column; *n/c*, nucleus cuneatus, at the top of the column of Burdach; *n/g*, nucleus gracilis or nucleus of the column of Goll; *cr*, restiform body, becoming the inferior peduncle of the cerebellum; the fibres in the latter can be traced in the figure, and need no explanation, except to say that fibre 2 represents the direct cerebellar column of the spinal cord. The course of fibres from the nucleus of the lateral column is not given. They go to the formatio reticularis and ultimately reach the parietal cortex. (From Bechterew.)

from the vestibule (which tells us of the body-balance, or its position in space). These fibres cross to the other hemisphere of the cerebellum. Lastly, a tract of fibres carrying impressions outward, known as the cerebello-olivary tract, which are axons of the cells of Purkinje of the *same* side, and end about the cells of the *opposite* olive.

All these fibres arborize about the cells of the sensory areas of the brain-cortex (especially about the cells of the ascending parietal, and many cells mingled with these in what has formerly been considered a purely motor region of the cortex), and these cells send off new axons which arborize about the large pyramidal or motor cells in the third layer of the ascending, frontal, or precentral lobe. These cells, you will remember, send off in a downward direction long axons which are the longest nerve-fibres in the body, and which ultimately end in arborizations about the motor cells of the anterior horn at the various levels of the cord. These fibres arising from quite an area, are gathered into a compact bundle, and are turned a quarter of a circle in the process, in order to pass downward toward the cord between the lenticular nucleus on the outside, and the optic thalamus posteriorly, and to the inside, and the head of the caudate nucleus anteriorly, and also toward the inside. When it has gained this new position between the nuclei it is termed the internal capsule, but while in the process it has a sort of fan shape, and it then is termed the corona radiata. The point at which the internal capsule bends about the apex of the lenticular nucleus is called the knee, and the extremities are called the anterior and posterior limbs. The anterior limb, except a very small portion next the knee, is occupied by fibres which coördinate the action of the frontal lobes, and also accommodates a bundle of fibres which run from the frontal lobe of the one side to the *opposite* lobe of the cerebellum. The extreme posterior portion of the posterior of the limb seems to be devoted to a similar bundle from the occipital lobes. The portion which is left, viz., a very small portion of the posterior part of the anterior limb, and the anterior two-thirds of the posterior limb, is devoted to the transmission of the motor fibres to the whole body, for it must be remembered that not only the motor fibres which descend in the cord are to be found here, but also those which furnish motor impulses to the cranial nerves and nerves of special sense as well.

These fibres have a definite arrangement in the capsule. Beginning a little in front of the knee and going backward, the order is as follows: eyes open, eyes turned, mouth opened, head turned (these two areas are just at the knees), tongue, mouth retracted, shoulder, elbow, wrist, fingers, thumb, trunk, hip, ankle, knee, big toe, toes.

From this comparatively wide expansion the fibres are gathered

into a still more compact bundle, and descend into the mid-brain, occupying that portion known as the crus. These motor fibres lie in the middle part of the crus, having on their *inner* side that bundle of fibres which was said to run from the frontal to the

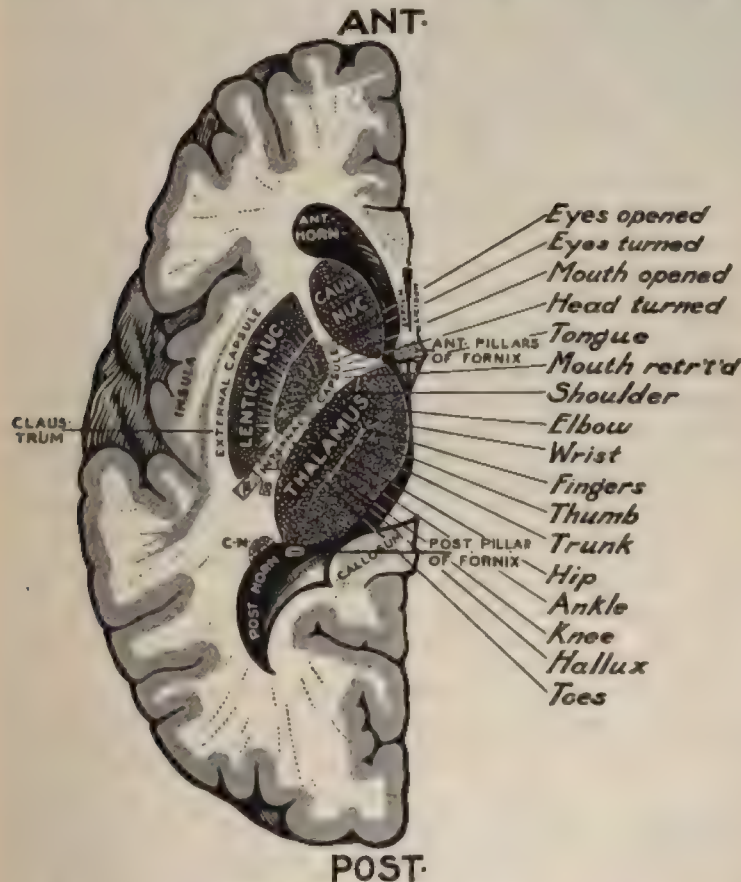


FIGURE 19.—THE INTERNAL CAPSULE.

The localization of the motor divisions is indicated by the text. The small square just posterior to the "toe" area indicates the location of the tract conveying ordinary sensory stimuli, the one directly behind that is the Optic tract, and the one lying toward the median line from this is the Auditory tract. (Gerrish, modified.)

other side of the cerebellum, and on the *outer* side is a similar bundle running from the temporo-occipital lobe to the opposite half of the cerebellum. These compacted motor fibres are now for the first time known as the pyramids. The pyramid now

enters the pons, and the fibres are again re-arranged into separate small bundles which lie on the anterior portion of the pons, and are separated from each other, as they descend vertically, by the horizontal bundles making up the middle cerebellar peduncle. On reaching the medulla, all the fibres of a side are again assembled into a compact bundle, now called the anterior pyramid of the medulla. They lie at the front margin of the medulla, but are capped toward the center of the medulla by the arcuate nucleus, and separated only by the anterior median fissure. Just behind them lies the fillet or lemniscus; behind that the inferior olives. This is its position throughout the medulla, but just as it enters the cord, at the level of the 1st cervical, it is divided into two unequal bundles, generally in the proportion of one to nine. The smaller portion, now known as the direct pyramidal tract, or the column of Turck, retains its present position throughout its whole extent, which is to about the middorsal region, rarely to 1st lumbar. It continually diminishes in size, since it continually sends off its fibres at right angles. These fibres pass through the white or anterior commissure, and arborize about the cells of the *opposite* anterior horn, and are supposed to have an inhibitory influence over the action of these cells. The other nine-tenths cross over to the other side of the cord, and take up a position near to the periphery, and just anterior to the posterior horns. This tract is now known as the crossed pyramidal tract. This crossing is known as the motor decussation. This column extends the whole length of the cord, but it continually diminishes, since it, as well as the direct pyramid, continually sends off fibres at right angles, which arborize about the motor cells in the anterior horn of the *same* side. These fibres convey tonus and voluntary motor impulses to these anterior horn cells.

The motor cells of the anterior horns send out axons which go out at the periphery of the cord singly, not leaving the cord in a compact bundle like that with which the posterior root enters the cord, and these fibres on the outside are gathered into a cord which unites with the sensory fibres just beyond the posterior root-ganglion, and at the point where it passes out of the intervertebral foramen which is hollowed out between the pedicles of the vertebrae. This combination of a sensory incoming (or centripetal), and an outgoing (or centrifugal) motor root is the type-form of all the spinal, and those of the cranial nerves that are called mixed nerves, viz., the 5th, 9th, 10th, and 11th.

These motor fibres now go to such parts as they furnish with motor impulses, and end in tendon, and muscle, and viscera, and vessels of all kinds, terminating there in the end-plates peculiar to the locality and the structure. Nerve-fibres never unite with others, nor terminate in another, but run from the motor-cell in the anterior horn to some form of separate distribution in the

structure which they are to energize. As the fibre approaches its destination it divides dichotomously (by twos), always at a node of Ranvier. In a voluntary muscle it goes to the sarcolemma, and terminates in a knobbed mass having from six to ten nuclei. In involuntary muscles the fibre passes through the muscle nucleus, and then breaks up into a plexus about the muscle-fibre. In glands, the branched terminals of the motor-fibre end in the nucleoli of the glandular cells. In tendons it ends in numerous leaf-like expansions. The sensory-nerves gather impressions from their end-organs, or inter-epithelial arborizations.



FIGURE 20.—MOTOR OR GANGLION CELL.

From the anterior horn of the spinal cord of a human foetus, 30 centimetres in length. A collateral branch (Golgi) from the axis-cylinder, or neuraxon, is seen; indicated by the star. Highly magnified. (Lenhossek.)

On review, it will be seen that motor nerves go directly from the cortex to the motor-cells in the anterior horn, and the new axons from thence directly to the end-plate or terminal in the structure which it is designed to innervate, while the sensory nerve terminates in the posterior horn, or in some nucleus in the cord or medulla, and that the new axon is soon interrupted by some nucleus. This indicates that a spinal motor tract is a device to convey impulses to the motor-cells of the anterior horns from the cortex, which is equivalent to saying that this impulse is sent to the anterior horn cells in response to the will. The motor-cells of the anterior horns stand always ready to obey this class of impulse and also any other stimulus that may be brought to them. We

have seen that at every level some of the fibres of the posterior roots arborize about cells in the posterior horn, from which new axons go up to, and arborize about, the motor-cells of the anterior horn. They are just as competent to release the pent-up energies of these motor-cells as are the stimuli borne to them from the brain by the fibres of the crossed pyramidal tracts, so we see that an action may result from a thought of the brain (volitional stimulation) or from a peripheral irritation brought in by the sensory nerve (reflex stimulation).

Every motion performed by the body falls within one or the other of these categories. The cerebellum is nothing more than an especially elaborate nucleus where impressions are re-arranged, and automatically modified, without the intervention of conscious thought. We see therefore that a movement may result from a desire to perform it—volitional. It may be systematized by the cerebellum after an initial impulse from the brain. Such motor responses are called automatic, and are the result of some education of the structures. Dancing, or piano-playing are good examples of this sort of reflex action. The last class is where a peripheral stimulation from a part is carried by the most direct course to the motor-cells for that part, and evolves the desired motor response. This is purely reflex action, and accounts for some of the so-called reflexes which we use in the detection of disease of the nervous system. We are not far wrong if we consider all motion to be a reflex answer to peripheral stimulation, and brought about by the mechanism of the reflex arc, the simplest one being the sensory nerve to the cell of the posterior horn, thence to the motor-cell of the anterior horn, and thence to the motor end-plate by way of the motor part of a mixed nerve. The next one is where we take in the cerebellum, and the most complicated when the cortex of the brain is involved, but it is just as truly a reflex arc as in the first instance.

GENERAL SYMPTOMS OF NERVOUS DISEASE.

If the nervous system is diseased, there will be disturbances of motion and sensation, and in the various phenomena arising from the imperfection of reflex action. Such losses will result in paralysis, or changes in the reflexes, disturbance of the control of the sphincters of the bladder and the rectum, or alterations in gait or posture, or defective sensory appreciation of peripheral stimuli, or ataxia, or pain, or trophic disturbances. Generally there will be some combination of these losses, but some of them may exist alone. Paralysis is the complete or partial loss of power in a muscle or group of muscles. If it involves a whole limb, or a part of it, the loss is called a monoplegia. If it involves a vertical half

of the body, it is called a hemiplegia, but when all four limbs are involved it is termed a diplegia, carrying the implication that it is a double hemiplegia. This is also sometimes spoken of as a quadriplegia. These forms are generally the result of a cerebral or peripheral lesion. If the lower limbs are paralyzed, we say that the person has a paraplegia (of the parts below), and is significant of a spinal origin. Minor grades of paralysis are termed paresis.

The differential diagnosis between paralytic conditions is much simplified if we class them into upper and lower neuron palsies. The upper (or cortico-spinal) neuron paralysis is due to a lesion of the cells in the cortex or their fibres, anywhere in their course from the cortical cell down through the brain, mid-brain, pons, medulla and cord, until they terminate in brushes about the dendrites of the motor-cell in the anterior horn at some level in the cord. The lower (or spino-muscular) neuron paralysis arises from a lesion of the cell in the anterior horn of the cord, or of the motor-fibre at some point in its course from that cell to its termination in the motorial end-plate in the tissue which it innervates. No such distinction is possible in the case of the sensory neurons, since there is no such unbroken pathway from the peripheral tissues to the cortex. The broad distinction between palsies of the upper and the lower neurons lies in the fact that those of the first class deal with the volitional and inhibitory, and those of the second with the reflex pathways.

The upper neuron palsies have the following characteristics: the muscles are not completely paralyzed; all the muscles of a limb are affected to about the same extent; the limb is stiff (spastic); the muscles are rigid from unrestrained tonic contraction; they are over-sensitive to a slight tap (myotatic irritability); tendon reflexes are increased; sensory reflexes are often exaggerated; atrophy is absent, except such as would be expected to arise from the disuse of a limb (disuse atrophy); the muscles are not flabby, since nutrition is not much impaired; the circulation is somewhat deficient, and therefore the limb is somewhat cold and blue, and there may be some edema of the extremity; sensory disturbances are absent, or slight, since, if present, they arise from associated damage to the sensory tracts; there is no reaction of degeneration to electrical currents. Hemiplegia usually indicates that the lesion is in the brain, while paraplegia is the usual result of a lesion in the cord. Pott's disease, transverse myelitis, softening from thrombosis, hemorrhage or tumors of the cord, generally produce a paraplegia, but, if affecting the cervical region, will involve the arms also, and produce a quadriplegia. Primary lateral sclerosis, and syphilitic paraplegia are confined to the lower limbs.

The lower neuron type paralyzes the muscles fully at the outset; recovery is often impossible, or slow and imperfect; the com-

plete type of paralysis is soon followed by a selective form in which single muscles, or groups of muscles, are the sole seat of the paralysis; the limb is never stiff, but is limp and sways by gravity (flaccid); tendons and muscles are so relaxed that the joint-surfaces become separated; myotatic irritability is absent; the tendon reflexes are lost; atrophy is complete and rapid; there is the reaction of degeneration; the circulation is always impaired, as the vessels are relaxed, the blood-pressure is reduced, and the capillary circulation is slowed; the limb is colder than in the first form, it is blue, and, while it sweats at first, remains cold to the end; sensory disturbances may not be present. This form is found in anterior poliomyelitis, both acute and chronic, amyotrophic lateral sclerosis, myelitis, either localized, or extending through the cord, syringomyelia when it invades the anterior horns, tumors and hemorrhages within the cord, softening due to thrombosis and embolism, and is also present in neuritis. The diagnosis of the site of the lesion whether it be a neuritis, or a damage to the cord, rests upon the distribution of the resulting paralysis, and the sensory losses. Muscle-groups, and not special muscles, are represented in segments of the cord, while definite muscles are innervated by nerves whose destination is well known. The anesthesia from a segmental lesion is of a part or section of a part, while from a nerve lesion it involves definite small areas. Lesions like amyotrophic lateral sclerosis may involve both the upper and lower neurons, and then the type of paralysis is not sufficient for a diagnosis. We must here investigate the order of the appearance of the symptoms, since at some stage of the disease it will be purely of one or the other type. General inflammations of the cord will always disturb reflex action, and the action of the bladder and rectum, and produce marked atrophies. While therefore some parts of the body may show the symptoms of one type, and another part those of the other, the disease will finally involve all parts in a paralysis of the second, or lower neuron, type.

Injuries to the spinal cord by fractures, or injuries of the vertebrae, may crush the cord, or completely sever it. There is a diagnostic difference in the resulting conditions.

If the cord is partially severed, there will be total paralysis, which may be asymmetrical; below the site of the injury, rigidity of limbs; increase in the reflexes; disturbance of the sphincters, either causing retention, or spontaneous evacuation; probably some tympanites.

If the cord has been completely severed, there will be total paralysis below the point of injury; the limbs will be relaxed, and not rigid; the reflexes will be abolished; the paralysis will be symmetrical on both sides; retention of urine always; tympanitis; vaso-constrictor paralysis, with the result that there is a dilatation of the subcutaneous veins, leading to an increase of the tempera-

ture of the part, and to priapism; increase in the genital reflex (tenderness of the testicles to pressure); loss of sensibility to pain and temperature, and usually to touch also, at levels corresponding to the segments injured. In the absence of these symptoms, we may conclude that the cord is not entirely severed.

LOCALIZING OR FOCAL SYMPTOMS OF DISEASES OF THE BRAIN.

Lesions of the brain produce certain symptoms by which we are able to fix the site of the damage, in addition to the general symptoms before stated, which denote that the brain has been injured, but give no clue to the particular structures which have been compromised. These localizing symptoms are both motor and sensory, of which the former are by far the most valuable, since, while there is a pathway from the periphery to the sensory cells of the cortex, it is by no means the simple linkage which we encounter in dissecting the route of motor impulses. Sensory impressions meet so many cell aggregates in their upward course, in each one of which there is a possible modification in direction or character of impulse, that the final destination, and certain route, are as yet somewhat matters of conjecture.

The possibility of localizing a motor or sensory lesion in the brain depends upon the following facts:

1. That the brain is that part of the nervous system in which we originate voluntary movements. Therefore if a member of the body is capable of motion from some peripheral stimulus, but refuses to move in response to the will, we know that the neuron whose cell is in the brain is injured in some portion, and some added data will often definitely localize the site of the injury. Since in a definite area of the cortex is localized the cell-area capable of inducing every known movement of which the body is capable (for the cortical areas represent movements, like grasping for instance, and not separate muscles), the paralysis of a single member, or part of it, shows us, if the reflexes are exaggerated (the mark of a lesion of some part of the upper motor neuron), that the lesion is cortical. Monoplegia, with increased reflexes, is therefore the mark of cortical damage. Since the fibres from the widely distributed cells of the cortex are gathered fan-wise, to appear as a small bundle in the internal capsule, the amount of one side of the body which will be paralyzed by a lesion of a given size increases as we pass from the cortex to the capsule. When the capsule is reached, we find, therefore, that the type is hemiplegic, instead of monoplegic.

2. The intellectual processes are the result of the comparison and correlation of impulses from many parts of the brain, and

when the process is completed, the act results from the stimulation of the area in which is stored up the muscular memory of the desired act. These are localized in different areas, and if the tracts leading to those areas are cut off the motion will not ensue, since the appropriate centers cannot coordinately be stimulated. Speech is an instance of such a grouping of specialized centers, and in the article upon aphasia the effects of various lesions are described.

3. The motor tracts in their downward course are brought into close relations with other structures, and the site of a lesion below the internal capsule causing a motor loss can be ascertained by a consideration of the associated losses.

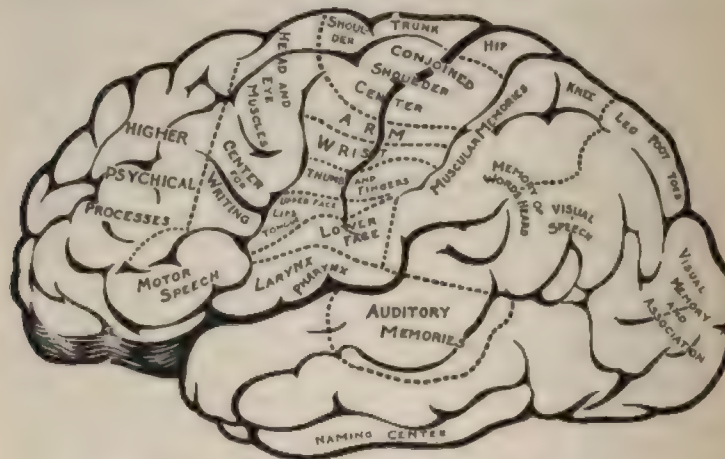


FIGURE 21. -LOCALIZATION OF THE CORTEX OF THE BRAIN.

SENSORY LESIONS. The cells for the appreciation of general sensation lie mostly behind those of motion, but are not absolutely isolated from them. The motor-area has some sensory cells, but the proportion decreases as we approach the parietal cortex, which is purely sensory. It may be said with a fair amount of definiteness that the cells for sense-perception are located in the middle third of the posterior central convolution, and the inferior parietal lobe, and that the face-area is in the lowest, and that for the leg is nearest the vertex. The theory of Ferrier and Mills locates them in the hippocampal region, but this is from experiments on animals, and it does not apply to man. This localization applies to all varieties of sensation, touch, muscle-sense, heat, cold, and pain. These several impressions are received by separate cells, so that one set appreciates touch, another the position of the limbs (this seems rather localized in the central lobes), another pain,

another heat, and still another cold, but stereognosis (the appreciation of the contour, weight, shape, and peculiarities of surface of an object) requires the perfection of all of these. When, therefore, any one of these fails, the individual will suffer to some degree from astereognosis. The fact that there is a sensory loss will not in itself be localizing, but since, as I have said, parts of the body are localized on the cortex, we may assume that the paresthesia of a single part is the result of a cortical lesion. The sensory fibres are gathered into a bundle, which passes in the posterior third of the posterior limb of the internal capsule, therefore as the motor loss from a cortical lesion was a monoplegia, so a similar sensory loss is the paresthesia of a single part, and as the motor loss from a lesion of the capsule, or tracts within the brain, was a hemiplegia, so from a sensory lesion in a similar location we may get a hemi-paresthesia, but not so certainly, since sensory representation is much more bilateral. As has before been said, destruction of areas or tracts produces paralysis of motion or sensation, while irritation of them produces exaltation of function, either muscular spasm, or pain in some degree; it may be severe pain in the other half of the body. The usual result of injury to the sensory cells is a paresthesia rather than an anesthesia, in the form of a slight numbness or tingling in the extremities, hands, and feet, and lips. Whether it be a decrease or an increase, it fades away into areas of normal sensibility. This differs from hysteria in just this indefiniteness of boundary, and also that there is no accompanying paralysis, and is often associated with a concentric diminution of the visual fields. While the loss is possibly for all varieties of sense-perception, it is often a dissociated one, and mostly of touch, and appreciation of the position of the limbs. The paresthesia from a cortical lesion is never absolute, and while it may be from the organic damage from a hemorrhage, or the pressure of a tumor, it may also be functional, even if there is damage of some sort in the vicinity. It is a frequent warning of an attack of Jacksonian epilepsy, or of idiopathic epilepsy, and often is circulatory from temporary arterial spasm, as well as from the more serious disturbance which culminates in hemorrhage or thrombosis. A person may be very ataxic with all the other sense-perceptions perfect, or suffer from an anesthesia or paresthesia with no ataxia, it may be with motor-palsy, or it may be the only symptom of cortical damage. It is never total, since sensory representation is more bilateral than motor representation. We know nothing of the sensory losses from lesions of other parts, and large areas of the motor area may be cut out without sensory losses of a permanent nature. There is a peculiar loss found in lesions of the motor zone, astereognosis. It may be absent at first, and present later; it is often found in persons whose hemiplegia dated from a period when memory

pictures had not yet been firmly fixed. Some think that the loss is due to the loss of one element of perception from the many necessary for its full perfection, while another view is that it is due to a loss of the power of fixed attention. The special senses have a similar definiteness in cortical representation. Sight is represented in the cortex of the cuneus, hearing in the first, and part of the second temporal lobes, while smell and taste are localized in the limbic lobe near the inside of the tip of the temporal lobes.

SUBCORTICAL LESIONS. If we could fancy ourselves looking at the under surface of the cortex of the brain, we should see a maze of nerve-fibres, tending downward from all parts of the hemisphere, to finally appear as a narrow band between the lenticular nucleus on the outside, and the optic thalamus to the inside posteriorly, and the head of the caudate nucleus to the inside anteriorly. This band is the internal capsule, and these are the neuraxons in their distribution to all parts of the body. These fibres rotate somewhat in their course, and appear in the capsule arranged as pictured in the figure. Passing still downward, the motor fibres appear as a yet narrower band in the foot of the crus, and thence they pass into the pons. Here they are spread out somewhat widely upon the anterior aspect, passing through a sort of reticulum made by the interlacing of the fibres of the two middle peduncles of the cerebellum. Proceeding from the pons, they are again gathered into a bundle called the anterior pyramid, on the anterior aspect of the medulla. At the lower border of the medulla, the anterior pyramid of the left side crosses to the right, and the right pyramid crosses to the left in a similar manner, forming what is known as the motor decussation. After this crossing, the motor tract assumes a position just anterior to the posterior horn in the lateral portion of the cord, and is now known as the lateral tract, or the crossed pyramidal tract. It is called the crossed pyramidal tract in contradistinction to about one-tenth of the original anterior pyramid of the medulla, which in most cases does not cross, but continues downward in the anterior part of the cord on the same side of the center line which it formerly occupied in the medulla. After leaving the capsule, the tract is always in close proximity to cranial nerves or their nuclei, or to the cerebellum, and therefore a lesion of the tracts might reasonably be expected to damage some other structure, and vice versa. The accessory damage therefore furnishes the grounds upon which we localize a lesion of the motor tract below the internal capsule. Before passing to these lower lesions it should be pointed out that the fibres are so concentrated in the capsule that a small lesion will produce a damage of much greater extent than one of the cortex, or one between the cortex and the capsule, while the fibres are still more or less widely fanned out in the corona radiata. In the capsule a lesion must have the

very unusual size of a pea to produce a monoplegia. Such a result is possible, but in the highest degree improbable, and the usual result of a lesion of the capsule is a more or less complete hemiplegia. A monoplegia might possibly result from a very small lesion in the crus, or the upper part of the pons, but, since the fibres are spread out in the lower part of the structure, a lesion of the same size in the lower part of the pons would result in a paralysis of more limited area. Lesions of the central ganglia are more sensory than motor, and resulting symptoms of both varieties will be discovered at the same time. The site of a lesion in the capsule will be shown by the portion of the body which is the seat of actual paralysis when the temporary general paralysis has passed away.

The sensory fibres pass through the posterior portion of the posterior limb of the internal capsule, as is shown in the figure on the preceding page. In this area the fibres of special sense are massed in bundles which lie toward the anterior portion of the sensory area. These bundles are from both hemispheres, and a lesion must be in the capsule or the cortex to produce any permanent losses. If the sensory fibres are injured in the internal capsule the loss or change applies equally to all forms of sense-perception, which aids us in localizing the lesion, since lesions in the cortex, pons, or medulla, cause dissociated losses, i.e., one or more varieties of loss, while another will remain normally acute. A finer localization applying to any level is sometimes possible in the case of slowly developing lesions. If motion is paralyzed, and, later, sensation also, we know that the common cause was first in the motor area, and had extended in the direction of the sensory tracts, or the opposite condition might be the fact. Hemianesthesia is generally due to a lesion in the posterior part of the internal capsule, or where the fibres are mixed with the motor ones, and is usually attended by hemianalgesia, or hemithermo-anesthesia. It is rarely absolute, on account of bilateral representation. As a rule the entire opposite half of the body is anesthetic to some degree after a sensory lesion in the capsule. By as much as a lesion lies above the capsule, by so much is it less competent to produce a hemianesthesia, and in the centrum semiovale there is apt to result an anesthesia of one limb, or part of the body also. The same losses, exaltations and perversions of sense-perception may be produced by lesions of the sensory tracts, although there is no single, long, uninterrupted tract like that of the motor system. We may find from any such lesion an anesthesia, or peculiar hyperesthesia, or both may be so combined that with an anesthesia the result of stimulation is a disagreeable sensation which may rise to a degree of pain if the stimulant is strong enough. The skin and mucous membranes anywhere may be the seat of this perversion, although some say that the cornea is

spared. There are perfect sensory reflexes in the cord and ganglia, all the way from the lowest portion of the cord to the cortex. If this sensory perversion is present, there will therefore be reflex symptoms of irritation shown in cries and reflex movements, designed to remove the part from the irritant which would be present if the organism were in a state of health. The respiration and pulse may be quickened, and the face may show distress. This is assumed to be reflex, since all the phenomena will be present, if the brain has been previously removed. It is not necessary that the sensory cells should be injured. An injury to the tracts will exalt or depress these sense-perceptions according to the degree of injury.

CRUS. The peculiarity which localizes lesions in the crus arises from the fact that the 3d nerve (motor oculi) comes out from the inner side of the crus, and the optic tract lies upon it. A lesion in this locality which is extensive enough to cause a hemiplegia will in all probability involve the 3d nerve, and also affect injuriously the optic tract. We should therefore expect with the hemiplegia a paralysis of the motor muscles of the eye (except the superior oblique, and the external rectus), and probably a hemianopsia. A lesion at this point gives rise to the syndrome of Benedickt and Weber. In this the eye on the side of the lesion is affected, and therefore the oculo-motor palsy is opposite to the paralysis of the face and body. The eye is turned outward, and cannot be turned upward, downward, or inward, the pupil is dilated, and there is ptosis, since the lid falls from the paralysis of the levator palpebrae superioris. The corpora quadrigemina are so near that they can hardly fail to be affected, and the red nucleus within the crus as well. If the posterior corpora quadrigemina are involved there is disturbance of hearing and of coördination, and the latter result will follow a lesion of the red nucleus. The sensory tracts, tegmentum, and fillet, lie just above the motor fibres in the crus, and they are generally involved. If they are injured, there will be an anesthesia of the opposite side of the body without any disturbance of the special senses, but if the acoustic fibres in the tegmentum are injured, there will be deafness if the lesion is bilateral. In lesions below the internal capsule there may be a loss of the perception of pain or temperature, accompanied by paralyzes of some of the cranial nerves, whose known point of origin localizes the injury causing the loss of these perceptions.

PONS. A small lesion in the upper part of the pons may be indistinguishable from one in the internal capsule, lesions in the lower part of the pons, only, being characteristic. A combination of paralysis of the limbs and body, with palsy of some of the cranial nerves is diagnostic of lesions in the pons and medulla. The lesion here is generally a hemiplegia, since the raphe is strong

enough to confine an ordinary hemorrhage to one side of the structure. If this is ruptured, a diplegia will be the result. Localization of a lesion in this locality is from the coincident paralysis of some of the cranial nerves. The 5th, 6th, and 7th cranial nerves run in this structure from the time that they leave their nuclei of origin, until they emerge upon its surface. The nuclei lie in the upper part of the pons. They are very liable to injury in any lesion of the pons. Crossed or alternate hemiplegia is especially a sign of a lesion in the lower third of this division of the brain-stem. This means that there is a paralysis of the face upon the same side as the lesion, and of the body upon the opposite side. The fibres from the brain to the facial nucleus lie upon the side of their origin in the upper part of the pons, but cross in the pons to gain the nucleus on the opposite side in its lower part, and therefore a lesion in the upper part results in an ordinary hemiplegia. If the lesion is in the lower part, the nucleus of the facial and its root-fibres are involved on the same side as their distribution, and therefore an alternate hemiplegia results. The nucleus of the 6th nerve (external rectus) is practically always involved, and therefore the eyes will turn away from the lesion, which is contrary to the rule when the lesion is higher up. If the nucleus of the facial is damaged, the eye cannot be closed, but if the fibres are injured on their way to the nucleus it can be closed. The pupils are contracted to a pin point. The tegmentum lies above or behind the motor tracts, and if the lesion is extensive enough to involve it, there will be anesthesia in the area of distribution of the motor palsy. The motor and sensory portions of the 5th nerve lie here, and may be involved separately, or in combination. If the sensory fibres are alone affected, we shall find anesthesia of the face on the side of the lesion, and a paralysis of the limbs on the other, from an implication of the motor tracts, but if the whole nerve is involved there will be the same sensory loss as above, and a crossed motor paralysis. Spasms and convulsions occur when the pons is affected even in acute diseases. The middle peduncles of the cerebellum lie on the under side of the pons, and many pontile symptoms arise from a simultaneous involvement of this structure. If the middle cerebellar peduncle is invaded there are vertigo, and vomiting, and tinnitus is usually present. Deafness may occur, and if the lesion is irritative, and not destructive, there may be gyratory movements, and forced one-sided positions of the limbs in lying, which may be accompanied by flexion of the head and eyes to the same side, but not necessarily to the same side as the lesion.

MEDULLA. Lesions here compromise some one or all of the nuclei of the 9th, 10th, 11th, and 12th cranial nerves. If the lesion is unilateral, we shall find a crossed hemiplegia, so far as the function of these nerves is concerned, and dysarthria with hemiplegia

has been known to occur. The cerebellar tracts enter that organ through the restiform bodies, and these tracts are joined by fibres from the nucleus cuneatus and gracilis, and also from the olivary bodies. If therefore, there is a lesion of the lower part of the medulla, it will cut off the tracts to the cerebellum, and give rise to vertigo and loss of equilibrium, but if above these tracts no such losses will exist. Clinically lesions of this area are so rapidly fatal that the theoretical results are not often seen. Owing to the close relation of the nuclei of both sides in the medulla, a small lesion usually produces bilateral symptoms. A characteristic lesion of the medulla is a labio-glossal-laryngeal paralysis, due to a very slowly progressive bilateral degeneration of the motor bulbar nuclei. It usually starts in the nuclei of the hypoglossal, next affects the spinal accessory, next the glosso-pharyngeal, next the pneumogastric, and occasionally the facial.

BASAL GANGLIA. It has seemed best to treat such lesions separately from others which are also subcortical, since all statements of a localizing significance must be made with many reservations. Most of the symptoms occurring in case of their injury or disease must be ascribed to a simultaneous injury of the tracts in the vicinity. The following functions have been ascribed to these ganglia, but somewhat inferentially. The caudate nucleus is supposed to have some relation to the movements of the legs, while the lenticular nucleus is similarly related to the movements of the arms, the act of eating, and the function of speech. The optic thalamus is known to be a sensory ganglion, and it has been claimed that every sensory tract ended in the thalamus before its ultimate radiation to the cortex. It is certain that a lesion of the pulvinar will cause only hemianopsia (a one-sided loss of vision), and this explains why the destruction of one thalamus produces no lasting anesthesia, since sensation has a bilateral cortical representation. A lesion of this body has been assumed to be the cause of a hemi-chorea, or hemi-athetosis, but both are now believed to flow from an irritant lesion of the neighboring motor or sensory tracts. When the facial muscles have been paralyzed to voluntary impulses they will often be found to respond to the calls of the emotions, while on the other hand, if the thalamus has been injured, the muscles will respond to volition, but not to emotion. From this it has been concluded that the proper response to the emotions depends upon the integrity of the thalamus. The same exhibition of causeless laughter, excitement and depression results from a tumor in the frontal lobe, and not from such a growth in the corpus callosum. The similarity in result of a lesion in the frontal lobe, and the optic thalamus, is explained by the discovery that a bundle of fibres originating in the cells of the frontal cortex passes through the anterior portion of the internal capsule, and ends in the external nucleus of the thalamus. The

person with a lesion in the thalamus cannot smell in the opposite nostril. Reflex thermic centers have been located in the basal ganglia, but not more exactly, as well as the vaso-motor, secretory, and trophic control of the opposite half of the body. Localization must be made by associated capsular symptoms.

Lesions of the external capsule and claustrum cannot be localized, but in some cases of paraphasia a lesion of those ganglia has been found in the left hemisphere.

CORPORA QUADRIGEMINA. These are visual and auditory paths, and the superior cerebellar peduncles contain the nuclei of the ocular nerves.

Lesions of the corpora quadrigemina alone are rare, as they usually are accessory involvements in lesions of the crus, or the anterior fossa. If the anterior pair were the site of a lesion, we should find oculo-motor palsy, loss of the pupillary reflex, and nystagmus. If the posterior pair were affected, hearing and coördination would suffer. A defective action of the oculo-motor nerve upon both sides is rare, and if it occurred it would point to a disease of the corpora; the same is true of the optic nerve, and if blindness is not a hemianopsia, nor due to optic neuritis, choked disc, or optic atrophy, it may be due to a lesion of the corpora quadrigemina. Both pairs are commonly affected in a discrete lesion, and a combination of characteristic symptoms would be diagnostic. Nothnagel says that there are only two focal symptoms which are absolutely diagnostic: 1st, a slowly increasing cerebellar ataxia, identical with that occasioned by that of the worm; and 2d, a gradually increasing, and not entirely symmetrical ophthalmoplegia, the superior and inferior recti being most often affected. Bruns says that the ophthalmoplegia most often precedes ataxia, by which it differs from a cerebellar lesion, where the ataxia is always first.

CEREBELLUM. Lesions of the cerebellum produce the following symptoms in the order of their frequency: Headache, vomiting, ataxia, asthenia, occipital headache, and tenderness of the neck with rigidity, an inclination to turn the body toward the side of the lesion, and convulsions. Because it sets up pressure on the pons and medulla, the latter particularly, there are some cranial nerve palsies from injury of the nuclei, polyuria, glycosuria, tremors and sudden death, these latter symptoms arising from pressure on the fourth ventricle. This organ preserves the equilibrium of the body, but has nothing to do with touch, pain, or temperature perceptions. The symptoms are practically those resulting from a reduction of the tonus to muscles when the lesion is destructive, shown by the asthenia, and by its irregular increase into spasm when there is irritation in the cerebellum. The faculty of coördination depends upon the perfection of function of the cerebellum, and when it is disturbed the coincident loss of

tone and of coördination produces paresis, ataxia, asthenia, incoördination, tremors, astasia (motor incoördination in standing). Lesions of the lateral lobes have been found post mortem which have produced no symptoms during life, but if the central lobe or worm is the seat of a lesion very definite symptoms are sure to result. The functions of the organ seem to be irritated by it as a whole, but one side also seems to be able to assume the function of the other. The prime requisite for the production of symptoms from the lesion of one lateral lobe seems to be that it shall be sudden in its onset, and rapid in its growth like hemorrhage, abscess, or tumor of rapid development. A sudden lesion of the lateral lobe results in weakness upon that side of the body, lack of coördination, and the patient is apt to stagger toward that side. In lesions of the cerebellar hemispheres, that a patient always staggers to the same side is of no great clinical value, since it is impossible to state whether the lesion is irritative or destructive. It is also found that movements cannot be reproduced with rapidity and precision, and the member soon tires. The reflexes are reduced, and the patient tends to bend to that side. Weakness of the ocular muscles upon the same side is found, and nystagmus results from an attempt to turn them to the injured side. Lesions of the head of the worm produce a tendency to fall forward, and of the back portion a tendency to fall backward. The staggering and ocular disturbances are accompanied by a vertigo which is increased on assuming an upright position. A tumor of the frontal region induces a similar vertigo, and an occipital headache, and in one-fifth of the cases a similar bilateral weakness of the reflexes. Irritative lesions are marked by an exaggeration of these same influences over the musculature of the body. There is muscular stiffness of the side of the body affected, and there is nystagmus in which the jerking is toward the side of the lesion, and the body is strongly arched to this side. When the middle lobe is affected in the same irritative manner, or both lobes are simultaneously affected, there is opisthotonos, or emprostotonos. If we have extension of a growth primarily in the cerebellum, but which later impinges upon other structures, the symptoms will relate to those structures secondarily affected. If on the motor tracts, we shall find spastic symptoms on the side opposite the lesion with increase of myotatic irritability, and a tendency to contractures. If it is on the floor of the fourth ventricle, the nuclei of the cranial nerves will be affected, and we may find paralyzes of the fifth, sixth, seventh, eighth, ninth, tenth, and twelfth nerves. The eighth is particularly apt to be affected, and we may get symptoms like those of Meniere's disease. Labyrinthine disease may closely simulate a cerebellar lesion, and septic processes in the ear may extend to the cerebellum. Irritation of the fourth ventricle may produce polyuria, and glycosuria.

Obstruction of the veins of Galen may set up a hydrocephalus, and sudden death results from pressure upon the nucleus of the pneumogastric.

DISTURBANCES OF THE SPECIAL SENSES:

VISION. The optic nerve is part of the brain, and diseases of the brain may produce blindness, hemianopsia (blindness of one-half of one, or of both eyes, and is homonymus if of both left or right halves, while it is heteronymus if it affects the left half of one, and the right half of the other eye), optic neuritis, choked disc, optic atrophy, or hemorrhages. Optic neuritis, choked disc, or papillitis, is that condition where there is an infiltration and swelling of the nerve-head, so that the clear outline of the nerve-head in the retina is clouded or blotted out. The nerve-fibres are normally so transparent that they pass out of the nerve and over on to the retina, without cutting off the outline of the circular aperture in the choroid through which the nerve passes. When they are infiltrated, they become translucent, and later opaque, and hide this margin, and the ophthalmoscopic image reveals a chrysanthemum-like body placed in this aperture. A steep prominence is thus formed, about two-third of a millimeter in height (refractive difference of two diopters), which is reddish, or reddish gray in color, and the arteries are contracted, and the veins are dilated. From the cells of the cortex at the cuneus, the visual fibres are gathered into the bundle known as the radiation of Gratiolet, and pass forward into the posterior portion of the internal capsule, thence into the pulvinar of the optic thalamus, thence into the external geniculate body, thence into the anterior corpus quadrigeminus, and, decussating partially in the chiasm, are distributed to the retina. A lesion of the cuneus produces blindness of a sector of a circle. This is a rather theoretical result, since clinically we usually find a hemianopsia if there is a lesion anywhere in the occipital lobe, and it may produce a simple dimness of vision. If the lesion is subcortical it is usually accompanied by symptoms of localizing value. If it is near the cortex in the left hemisphere we shall expect to find with it some form of word-blindness, or aphasia. If it is in the internal capsule, or near the optic thalamus, there is usually hemianesthesia, or hemiplegia combined with it; if in the external geniculate body, the fibres going to the anterior corpus quadrigeminus are involved, and the movements of the pupil are compromised, and we get Wernicke's pupillary reflex, which can only occur when a lesion is in the optic tract, or basal ganglia, and does not occur in lesions of the cortex. This reflex is, that if light is thrown into the pupil on the blind side there is no pupillary response. When a lesion is confined to

the anterior corpora quadrigemina there is no loss of vision, but the movements of the eyes cannot be ensured, and hence single vision is lost, and the person suffers from diplopia. This causes vertigo, and a staggering gait like that resulting from cerebellar disease will result if the red nucleus, lying under the quadrigemina, is also affected by the lesion. The lesion of an optic tract upon one side will cause hemianopsia, and presents Wernicke's sign also. Lesions of the crus will also cause hemianopsia associated with motor paralysis, oculo-motor palsy and ptosis. The result of lesions of the chiasm is described in the chapter upon cranial-nerve lesions. None of the lesions just described are competent to produce blindness of one eye, i.e., both halves, or a concentric diminution of the field of vision, but such conditions arise only from diseases of the eye itself, or of one optic nerve, and are visible with the ophthalmoscope, or are due to hysteria, and to be diagnosed by the results of a general physical examination. While persons learn through all the senses, some acquire knowledge more easily through one sense than through another, and so all are divided into visuals and auditives, according to that selective route of acquirement, yet it is a fact that the greater proportion learn through the eye than the ear. When therefore the cuneus is injured, especially upon the left hemisphere, the person often loses the power to recognize formerly well-known objects by sight, while the sound of the name may identify them to him. This is psychical blindness.

If the cortex of the angular gyrus, located at the junction of the occipital and parietal lobes, is injured, we get a modification of psychical blindness, since at this point are stored up the memories of words seen. This renders the person unable to read, and is called word-blindness (alexia). It is rare that such a lesion can occur, and not affect the subcortical tracts which lie just beneath. If these are injured, hemianopsia will result, and it may be accompanied by hemianesthesia, and hemiataxia, and if the lesion projects into the tracts running forward from the center for visual word-memories, the person may also lose the power of writing (agraphia). Alexia is therefore usually complicated by agraphia, hemianesthesia, and hemiataxia. The lesions of the visual cortex are naturally different in grade, and may be irritant, as well as of the destructive nature just dwelt upon. If the cortex in this region is irritated, the central stimulation will awaken memory images whose appearance would be ascribed to the accustomed peripheral stimulation of the retina. This central irritation gives rise to visual hallucinations, and is the probable cause of the hallucinations which are common in the delirium from all forms of intoxication, whether it be caused by alcohol, the poisonous metals, or from infections. They are unilateral as a rule, and may precede attacks of Jacksonian epilepsy, and constitute the aura

in these cases. They may be simple flashes of light or a play of colors, or may amount to definite pictures of some complexity.

DISTURBANCES OF HEARING. In the discussion of these disturbances the same difference will be shown between irritative and destructive lesions which were displayed in the visual areas. Hearing, as a special sense, is bilaterally represented, and therefore deafness will not result from a unilateral lesion of the first, and part of the second temporal lobes where this sense is cortically represented. If however there is irritation of the cortex in this locality, upon either side, the person will suffer from auditory hallucinations, and the irritant may be organic change in its early stage, or an intoxication of any form. This accounts for the noises and voices heard in delirium, and also for the auditory *aurae* in the various forms of epilepsy. If these lobes in the left hemisphere are destroyed, we get a peculiar symptom-complex, since here are stored up the memories of words heard, and of musical harmonies. When these are lost the subject can no longer recognize objects by their spoken names, and cannot understand conversation, and since we sound words to ourselves before speaking them, as if we must correct our conception by a central auditory test, the person soon becomes a mute. This is amnesic aphasia, and the person speaks a jargon without meaning. This loss is not always symmetrical, any more than are the losses of sense-perception, and therefore a person may lose the memory of spoken words, and still retain that for harmonies. The loss, when confined to the appreciation of spoken speech, is psychical deafness. Since reading and writing are subordinate to emissive speech, they are both liable to be impaired when there is an auditory loss. The amount of the loss is variable, and varies from the same lesion in different persons, according to the amount of their acquirements. The losses are in inverse order to their acquirement. A person who is word-deaf will fail to appreciate the meaning of sentences, while he may still appreciate single words. The names of objects acquired first will persist longest, and nouns will be retained after they have failed to recognize adjectives, verbs, and prepositions. After they have lost the use of nouns, they may still be able to make themselves understood by the use of round-about methods of description of their uses or properties. Occasionally all word-memories are lost. The area for the storage of word-memories is a little larger than that utilized for hearing alone. Deafness is rare from a subcortical lesion, on account of the fact of double representation before referred to. The cochlear division of the auditory nerve alone conveys sound impressions, and the fibres lead to the lemniscus of the same or opposite side, and the posterior corpus quadrigeminus. A lesion of one lemniscus cannot produce deafness (double representation again), but when both are affected deafness will result, and this has occurred in pontile lesions.

DISTURBANCES OF SMELL. Such disturbances are rarely seen except in organic lesions, and in mental disease. The cortical representation is supposed to be in the uncinate gyrus of the amygdaloid nucleus, which is located at the tip of the temporal lobe, and taste is also, by some, located here. Irritation of this area will cause hallucinations of taste and smell, and such an aura sometimes precedes epileptiform convulsions.

LOCALIZING SYMPTOMS OF LESIONS OF THE SPINAL CORD.

Paralysis is in itself a localizing symptom, since the segment from which any given muscle is innervated can easily be found in the charts for localization. If, therefore, a muscle shows paralysis that fact alone indicates the presence of a lesion in the muscle, nerve, or a definite portion of the central nervous system. A little further study will give grounds for a decision as to which one of the three just named is the actual seat of the lesion.

REFLEX DISTURBANCES. Tendon-reflexes suffer variation in two directions, viz., the reflex may be exaggerated, or it may be diminished or lost, and the changes have a localizing value. If it is exaggerated, it will be the result of a direct irritation of the cells of the anterior horn from a local cause, or from a general neurasthenic condition. If from direct localized irritation, the exaltation will be confined to a single reflex, and there will be weakness and some degree of atrophy of the parts innervated from the segment, or segments, essential to this reflex. The localization of the lesion is then a simple matter. If it arises from a general neurasthenic condition, the simultaneous involvement of all the reflexes, and the associated symptoms proclaim the character of the process. If it results from a lesion of the upper neuron, the parts affected will show a moderate degree of generalized paralysis, without marked atrophy, or conspicuous changes in temperature, and there will be some grade of rigidity in the limb. that localizes it in some part of the tract from the cells of the anterior horn, up to and including the cells of the cortex. We gain our knowledge of its vertical position from the fact that all the reflexes below the lesion will be exaggerated, while those above will be normal. The reflexes will be lost in coma, conditions of shock, and sometimes in the invasion period of febrile disorders. Their loss under these conditions has no localizing value. They will be lost when the limbs are rigid from fibro-tendinous changes in the muscles, and here have no localizing value. They will be lost in section, or complete degeneration of the sensory or motor fibres, or of both, of a peripheral nerve. The loss may be localized in the nerve

from the fact that if in the motor fibres alone there will be motor paralysis in the muscle to which it is known to be distributed. If it is in the sensory fibres alone, there will be sensory losses in areas

	Motor.	Sensory.	Reflex.
	Stern, mastoid.	Integument	
	Trapezius	Neck and head	
	Diaphragm	Neck and shoulder	
	Serratus		
	Shoulder	Shoulder	Scapular reflex
	Arm	Arm	
	Hand	Hand	
	Ulnar and below.		
	Intercostal Muscles	Anterior side of thorax	Epigast. reflex
		Pit of stomach	
	Abdominal Muscles	Abdomen (Umbilicus)	Abdominal reflex
		Posterior part: upper half	
		Pubes and scrotum anterior part	Cremaster reflex
	Flexors, hip	Outer	
		Thigh	
	Extensors, knee	Anterior	Knee jerk
		Inner	
	Adductors		
	Abductors	Leg, inner posterior lower half	Gluteal reflex
	Extensors?	Back of leg	
	Flexors of knee?		
	Leg muscles for foot	Limb and foot, except inner side	Foot or ankle clonus
	Perineal and anal muscles	Perineum and anus	
		Integ bet. coccyx and anus	Plantar reflex

FIGURE 22.—LOCALIZATION OF FUNCTION IN THE SPINAL SEGMENTS. (From O'Connor.)

of skin known to be innervated from this particular nerve. The reflexes will be lost if the tracts in the posterior columns of the cord have been destroyed. The sensory losses in skin areas, discovered as before, with no corresponding motor loss, will show

that it is in the cord, since, being in a tract made up of fibres coming from many different levels, the sensory losses will be in patches in many separated parts of the skin in the region below the site of the lesion. Reflexes will also fail if there is destruction of the cells of the anterior horn in one or more of the segments making up the motor part of the reflex arc in question.

It will permit us to localize it in the anterior horn from the fact that the sensory supply is still perfect, and we can select the definite segment from the fact that we have charts which point out the segment from which any given muscle is innervated. Loss of the reflexes will also occur when there is a hemisection of the cord involving one lateral half. We know that it is of one half of the cord from the fact that the side on which is the lesion will be paralyzed, while the opposite side of the body will be anesthetic, but the reflexes will be lost on both. This is the Brown-Sequard syndrome. The vertical level of the lesion will be revealed from the fact that the next segment above the site of destruction will be irritated, with the result that there will be a zone of hyperesthesia in its area of innervation, and from the charts the segment may be identified. (See page 67.) If the cord be completely severed there will be complete loss of the reflexes below the lesion. The reason for this loss in place of expected exaggeration, as is the result of the release of inhibition in partial losses, is not certain, but has been ascribed to loss of tonus.

Skin-reflexes are not well enough understood to be of as great localizing value as are the tendon-reflexes. They probably depend upon the integrity of definite centers in the cord, but their position is not known. Since they are not always present in health, and may be inhibited by volition, their value is still further diminished. They are never exaggerated by organic disease, but may be in general conditions which produce a condition of nervous irritability in the whole nervous system. They are never lost in hysteria, while they may be in organic disease, and it is a point in differentiation when that disease is in question. If the tendon-reflexes are increased they are commonly lost, and so furnish data for an opinion of the presence of organic changes, and in a unilateral lesion may give information concerning the side of the lesion. Babinski's toe-sign (over-extension of the great toe on stroking the sole of the foot) locates a lesion in the upper neuron, but will not define its vertical level.

Clonus may arise from a pretty severe lesion of the upper motor neuron, or it may arise from irritation of the motor cells in the anterior horns of the cord. If it arises from an upper neuron lesion it has no further localizing significance, but if from a lesion of the horn-cells, the clonus will be limited to the sphere of influence of that segment. It is then localizing to a definite level, and it also excludes upper neuron disease.

MUSCULAR SPASM. This is possible whenever the upper neuron is affected, but does not follow disease of the anterior horn-cells, or of the muscles. If we find that the limb becomes rigid on attempts at motion, sometimes when passive, we may deduce that there is a lesion somewhere in the upper neuron. This spasm may be alternately of the flexors and extensors, causing the limb to shake violently. This has been called spinal epilepsy, although not at all epileptic in fact. Vertical localization is only possible when certain higher levels furnish symptoms for exclusion.

FIBRILLARY TWITCHINGS. Here the whole muscle does not contract, but a few fibres here, and others there, make a flickering on the surface of the muscle, but may be numerous enough to disturb its balance. It never arises from disease in the muscle, and an irritant lesion of the upper neuron throws the whole muscle into spasm, and we therefore know that the cells of the anterior horn are beginning to degenerate, and from the charts we can discover the segment which is affected.

CONTRACTURE. This has a localizing value, if it is of a single muscle, or part, but as a rule only as a factor in the history of the case.

ORGANIC REFLEXES OR BLADDER AND RECTAL CONTROL. Lesions of both the upper and lower neuron will disturb the control of the sphincters of the bladder and the rectum, and therefore the localizing value is small. If however the reflex center in the cord is destroyed the detrusor will be paralyzed, the bladder will be over-distended, and finally overcome the power of the atonic sphincter, and cause a constant dribbling of the urine. This is called passive incontinence, and is somewhat significant of such a lesion. It may be of value to remember, that while retention and suppression of urine are common in hysteria, incontinence is not a symptom.

SENSORY DISTURRANCES. Sensation may be disturbed by a lesion of the structures to which the sensory nerve is distributed, by a lesion of the peripheral nerve, by a lesion of the posterior roots, by disease of the tracts and cell-areas in the cord, and at any point in their further course up to and in the brain. After we pass above a definite level in the cord, the localization is only possible by the study of the entire symptomatology of the case. If the loss is due to a local lesion of the surface, the reason of the disturbance is apparent. If it is due to disease or injury of the peripheral nerve, it will be for all varieties of sense-perception, and there will be an accompanying paralysis, or if the branch is entirely sensory, the definite area of loss will furnish the requisite data for its localization. If the nerve-roots are involved the fact is indicated by the character of the results, and the vertical localization is indicated by the area of distribution of these symptoms. Conduction of sensory impressions from parts below is impaired in an

increasing degree, and reflex action of the cord at that level is compromised. Pain is a prominent symptom, and is felt in any of the structures innervated from the given level. This pain may be felt in areas which have lost common sensation. Girdle-pains have their origin in this condition, as well as in more central lesions in the posterior columns, and are sensations of constriction in a more or less definite band about the body or a limb. If the fibres are affected immediately after their entrance into the cord, the result will be a loss of sense-perception in the parts below, and will generally be for all qualities, since at once the fibres for all sensation are gathered into a narrow band on the external surface of the columns of Burdach. This universality of loss will point to a localization of the lesion in the posterior columns, since the fibres almost immediately become widely separated in their further course to the tracts in which they ascend. Since sensation to any part is subserved by the associated action of at least three contiguous segments, localization must always include one segment above that designated as the one supplying any particular area. If we find that equilibrium or accuracy of motion (static ataxia, or incoördination) is exclusively affected we may localize the lesion in the direct cerebellar tracts (it is never there exclusively except from tumor), while if it is a loss solely of the perception of pain and temperature, we should fix the loss in the cells about the central canal, or in the antero-lateral tracts which are the upward paths from these cells. In any case we can fix the upper level alone of the lesion. Pain in all these cases, except from root lesions is localized in the areas of the distribution of the involved nerves, and never in the back, pain in which is caused by root lesions, or general neurasthenic conditions, or hysteria, and in the latter case is movable by suggestion.

LESIONS OF THE CAUDA EQUINA. These are like those of a mass of peripheral nerves, and so are segmental in distribution. The inner fibres alone suffer from the slighter grades of violence, and the outer also only when the injuries are more severe. To diagnose this lesion from disease in the segments the examination must reveal a physical disturbance of the vertebrae or sacrum, else we must conclude that the symptoms arise from slow compression from some internal cause, as a tumor for instance. If merely the lowest roots are injured, the lesion will be restricted to the coccygeal nerve, and the only symptom will be a paralysis of the levator ani, with anesthesia of the anus and perineum, the sphincter of the anus being intact. If the lesion is below the level of the second sacral root, the anesthesia of the skin is diagnostic, being saddle-shaped, including in its upper part the skin about the anus, perineum, and posterior aspect of the scrotum and penis, with a small anesthetic area running downward from the perineum along the posterior internal aspect of both thighs.

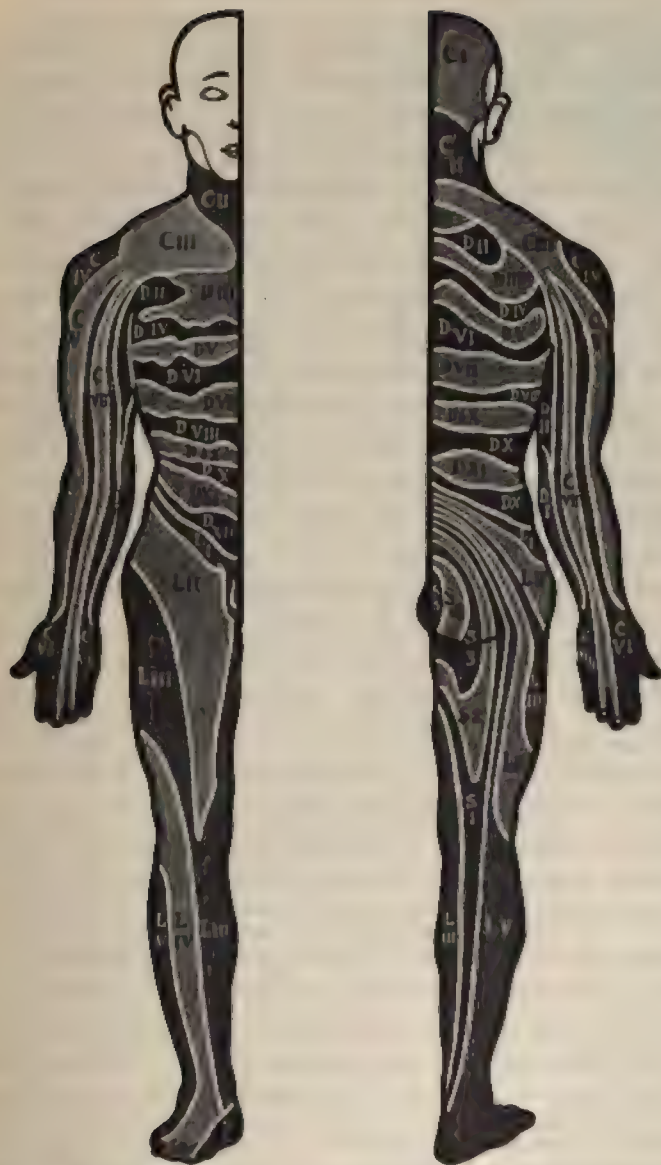


FIGURE 23.—AREAS OF INNERVATION OF THE SPINAL NERVES.

The numerals, with the letters, refer to the spinal segments from which these areas derive their sensory innervation. (Modified from Starr.)

The mucous membrane of the urethra and bladder will also be anesthetic. The bladder and rectum are paralyzed, and the anal reflex is lost. In both these and the preceding lesions there is no muscular paralysis of the lower limbs, and the knee-jerk and plantar reflexes are normal. If the lesion is below the third lumbar vertebra, the third lumbar roots escape damage, the anterior crural and obturator nerves will escape; the sciatic, gluteal, and pudic nerves will be involved. The muscular paralysis and atrophy will affect only the muscles supplied by the last-named nerves, and therefore there will be paralysis with wasting of the glutei and hamstring muscles, and of all the muscles below the knees. The anus and bladder will be paralyzed and there will be anesthesia in the corresponding root-areas, but the knee-jerks will remain normal. If the lesion is just above the first lumbar root there is paralysis with wasting of all the muscles of the lower extremities, anesthesia of the lower extremities, buttocks, perineum, and genitals, with the exception of the inguinal regions, and the upper and anterior part of each thigh. There will be a paralysis of the bladder and rectum, with loss of sexual power, and loss of the knee-and ankle-jerks, and of the sphincter and anal reflexes. If the whole cauda equina is involved, the paralysis will be that just described, but the anesthesia of the lower extremities, buttocks, and genitals will be complete.

GAIT. The gait of a patient is often diagnostic so far as concerns its classification as paralytic, spastic, or ataxic. The paralytic gait is produced only by a lesion of the lower motor neuron, but mere inspection of the gait will not certainly reveal the closer localization of the lesion. This gait may result from a lesion in the anterior horn, or of the peripheral nerve, and will have the accessory symptom of rapid wasting of the muscles. In both these cases the whole limb must be dragged along, and if typical, the patient will be obliged to make use of crutches or a cane. If the lesion is in the cells of the anterior horn there will be a preliminary paralysis of the whole limb, which will be followed by a definite paralysis of a group, or of several groups, of muscles. All the muscles of the limb may be paralyzed permanently if the lesion in the cord is sufficiently widespread, but clinically the condition is rarely so extensive. In this case the articulations are relaxed, and the joint surfaces become separated, the feet turn in, and the big-toe is scraped along the ground. The marks, then, of horn-lesions are paralysis of the function of the limb as a whole, with flaccidity, marked atrophy, separation of joint surfaces, inversion of the foot, and scraping of the toe on the ground. If the peripheral nerve is affected, the limb as a whole may not be paralyzed, but the flaccid paralysis of a part may render it just as useless as an implement of progression. The common seat of such a lesion is in the anterior tibial, which results in "drop-

foot." So far as the peroneal region is concerned, the phenomena are the same as just described. There is a flaccid paralysis with rapid atrophy of the parts involved, and there is relaxation of the joints. In this case, the fact that the peripheral part of the limb is paralyzed, while the proximal part is competent, produces a characteristic gait. The upper part of the limb can be extended or flexed, while the lower part hangs loosely. In walking, therefore, the upper leg is lifted high enough to allow the dependent foot to be swung clear of the ground, and this accentuated flexion of the thigh produces what is known as "steppage gait." If the lesion is in the upper motor neuron we have weakness, but not actual paralysis in the limb affected until a late period of the disease when the patient is helpless, and the variety of gait cannot be a factor in diagnosis. Since any lesion in the upper motor neuron releases the spinal reflexes from the inhibitory control of the brain, the tendon reflexes are all exaggerated, and the limb becomes stiff (spastic). Since the trophic centers lie in the anterior horns of the cord, and not in the lateral tracts, no atrophy results, except such as would be expected to arise from disuse. Two classes of cases are found in this category, viz., those arising from lesions of the tracts, and those from lesions in the brain. In the first case the stiffness of the limb makes the complicated process of walking unusually difficult, since that action is properly performed only when we are capable of instantaneously making numerous slight muscular readjustments at every stage of the process. This is further complicated by the fact that the limb is weak. In the study of the physiology of the cord it was stated that the stretching of a muscle when inhibition was removed from the cord would produce clonus, and we therefore have a weak limb which is stiff, and the muscles of which are the seat of possible clonus. All these factors coöperate in producing the peculiarities of the spastic gait. The leg being stiff tends to scrape along the ground "en masse," and to avoid this as far as possible the pelvis is tilted up on that side; the leg is swung forward as one piece; since it is weak the step is short, and the propelling leg is the seat of clonus, so soon as the muscles are put upon the stretch. This makes the posture of the person uncertain and teetery; this is called "titubation." Observe for future reference that the steps are placed in a straight line, and that they are short. In the second case, where the cortex is the site of the lesion, the adductor muscles in the thighs are spastic to a variable extent, sometimes to such a degree that the knees rub together, or so much that the legs cross, and we get "cross-legged" progression, or "scissors-leg." If now the lesion which has produced a pathological gait is not in either the upper or lower motor neuron, but is in the posterior columns of the cord, or the direct cerebellar tracts, or cells of the column of Clarke, we find an ataxic gait. There has been an injury of the fibres which

convey sensory impressions to the reflex arc, or data as to the tension on tendon and muscles, or pressure upon joint-surfaces to the cerebellum. We stand steadily or walk correctly from the ability to constantly readjust our position by the information conveyed to us from sensory impressions from the soles of our feet, and knowledge of the pressure and tension that is being exerted. The results of such losses are these. The muscles are not paralyzed, but force is exerted without proper direction unless guided by vision. The leg is lifted extravagantly, and the foot is projected without accuracy of direction, or to an equal extent in successive steps. It is not set down with precision, but is slapped down, the heel usually striking first. The result is that the person sways from side to side, the steps are of irregular length, and are not in a straight line. The feet are widely separated, both in walking, and in standing, and walking backward accentuates the disability.

SCHEME OF EXAMINATION OF CASES OF NERVOUS DISEASE.

Name—Sex—Age—Civil Condition—Married—Single—Widow or Widower—Resides—Date.

FAMILY HISTORY. Father. Mother. Condition of their health and of their ages, and their history in relation to habits and diseases. If either or both are dead, find age at death and cause of death. Number of brothers and sisters, and the patient's place in respect to the age of the others. Their state of health, ages, and if dead, cause, and age at death.

PERSONAL HISTORY OF PATIENT. Habits, present and past. If married, the state of health of the husband or wife, the number and health of the children, and miscarriages, if any, and their relation to living children. Patient's manner of birth. His health as an infant, convulsions if any, and the cause assigned for them. Diseases of infancy, and their severity, and after-effects. Diseases of childhood with their grade of severity and after-effects. Diseases of adult life with particulars (venereal especially). Injuries suffered.

PRESENT ILLNESS. When symptoms first appeared and what they were. Order of onset, rate, and if preceded or accompanied by fever or pain, and what cause was assigned for the onset. Present symptoms complained of. This includes all disabilities or abnormal sensations, or sphincter disturbances which the patient is able to note. (These are subjective symptoms, if they are such as can only be perceived by the patient).

EXAMINATION. General condition of nutrition, and size and weight. Marks of injuries or diseases.

Manifest marks of degeneration.
Firmness of muscles, and atrophies, or hypertrophies.
Ability to perform movements.
Possibility of passive movements.
Movements of eyes:
 Hemianopsia.
 Extent of visual field.
 Extent and anomalies of color field.
Movements of face.
Movements of tongue.
DEEP REFLEXES:
 Knee-jerk.
 Achilles-jerk.
 Ankle-clonus.
 Wrist-jerk.
 Jaw-jerk.
 Malar-jerk.
SUPERFICIAL REFLEXES:
 Plantar reflex.
 Babinski's reflex.
 Cremasteric reflex.
 Hypogastric reflex.
 Epigastric reflex.
 Pharyngeal reflex.
 Corneal reflex.
LOOK FOR:
 Argyll-Robertson pupil.
 Romberg's sign.
 Kernig's sign.
 Hypotonia.
 Tremor.
 Incoördination.
 Anesthesia.
 Hyperesthesia.
 Analgesia.
 Thermesthesia.
 (These are objective symptoms).
 Orientation. (Objective and subjective).
 Sphincter disturbances. (Organic reflexes).
MENTALITY:
 Aphasia. What variety and extent.

The methods by which the definite senses are examined are stated in detail in the description of the various conditions in which they are found to be perverted, and it is necessary here to speak only of general methods of investigation. Tests for the cutaneous sensibility may be made by ascertaining whether or not

the patient feels the touch of a camel's-hair brush, or of a little pledget of cotton, or still better if the light touch of the examiner's finger is perceived. The latter is the better instrument, as it is always accessible, and with a little practice the touch may be very delicate, and the relative sensitiveness of various areas is capable of very accurate estimation. Aesthesiometers of many varieties are for sale at the instrument-makers, but are not usually a necessity. The perception of pain may be investigated by pricking the surface in various areas with a common pin, and this instrument is better than a needle, since the latter may be so sharp that its entrance is comparatively painless. The perception of temperature is to be tested by the alternate use of a test-tube filled with cracked ice, and another filled with hot water. Care must be used that the cold tube is kept dry on the surface, else tactile sense may discern the moist surface of the one as opposed to the dry surface of the other. In the prosecution of all these tests the eyes should be carefully covered, and the site of the tests should be abruptly changed, and not only the kind of sensation, but its localization should be required of the patient. Response should be prompt, since delay of perception is a grade of loss. Remember that in temperature investigations a patient may lose the perception of heat, and not of cold, and vice versa. I have found it necessary to warn patients that no restraint is demanded of them, for in some cases the tests for pain gave at first confusing results, as the patient thought it the part of valor to avoid any symptoms of distress as long as possible. Tremor is tested by showing the teeth with as much rigidity of the lips as possible; by opening the mouth and thrusting out the tongue as far as possible, while at the same time the arms are stretched to their full extent above the head; by stretching out the arms at full length in front of the body, and at the same time separating the fingers. A more delicate test of tremor of the hands is by letting the tips of the outstretched fingers lightly press against the palm of the hand of the examiner. A very faint and fine tremor is discernible by this method. Handwriting will show tremor and ataxia also. The detection of passive and intention tremors is described in detail in the articles upon paralysis agitans and multiple sclerosis respectively. Ataxia and incoördination are stated in full in the article on tabes dorsalis. As a basis for diagnosis, the first question to be decided is this: Is the disease so sudden in onset that it must have resulted from traumatism (hemorrhage for instance), or gradual enough to allow an inflammatory process to be the cause, or has it been slow enough to have been the result of a degeneration? Is the locality of the lesion in the peripheral nerves, or is it central, i.e., in the spinal cord, or brain. If it is located in the central nervous system, is it upper, or lower, neuron in type?

THE THERAPEUTICS OF NERVOUS DISEASES.

The therapeutics of diseases of the nervous system differ from that of the body in general for several reasons. The medicinal treatment of the diseases of the rest of the system has been a slow accretion of the experience of the centuries in its quest for a healing agent for pathological changes in material structures which have been more or less possible of demonstration to the senses, or have been so readily influenced by remedies that numerous experiments have been possible, and the results have been unequivocal. Nervous diseases have not rested upon changes which were palpable or appreciable, some of them have not even yet yielded their secret to our more refined methods of investigation. They, in general, are not self-limited, and some of them take years for their full developement, and hardly one of them is curatively influenced by any kind of medication with enough rapidity to make the results striking, or even appreciable, except by observation for a long period. It may be more explicitly stated that until within a very few years, the causes and the seat of the characteristic pathological changes pertaining to any special disease were not known at all, and some are still shrouded in the same obscurity. All schools of medicine were therefore unable to furnish much material for our therapeutic guidance, since they were generally in no position to identify, or to rightly interpret the significance of the symptoms concerning which they were making their reports, and which they were assuming to have been relieved by their medication. This statement is written in full knowledge of the claims of both schools, and of the therapeutic accumulations of the years before even a few of these diseases could be diagnosed with any degree of accuracy. We may own that many a physician has cured a complaint which he could not diagnose, but we cannot accept his therapeutic conclusions, when we are certain that he was ignorant of the identity of the symptom-complex for which he announced the discovery of a remedy. We may perhaps infer that his description refers to the disease which we have under consideration, but we must repeatedly obtain similar results in order that his statements may carry conviction. Therefore it may be safely affirmed, no matter what may have been the school of medicine, or what may have been the nationality of the writer, that any remedy recommended over thirty years ago, for any given nervous disease, should carry very little weight, for no one at that time could know either the etiology, pathology, or symptomatology of the disease for which he makes the recommendation. When one looks over the chronology of the discovery and primary isolation of the actual changes characteristic of the

most common diseases of the nervous system he is struck by their juvenility, since dates like 1868, 1870, 1875, and so on, are the rule, and, as I have said before, many of them still hold the secret of their cause and its pathological expression. Who can certainly tell the cause of Epilepsy, of Chorea, of Hysteria, or even of Tabes Dorsalis? In many cases therefore symptomatology is the only possible basis for prescription and, comparatively speaking, is the only scientific method. The validity of any method however is nullified in organic diseases by the fact that while nerve-fibres may be regenerated, nerve-cells once destroyed are never replaced by tissue capable of nervous function. If therefore we have grounds for belief that the disease has destroyed nerve-cells, we know that medicine, however skilfully selected, and by whatever method, can do no more than stimulate the remaining cells to their highest grade of function. All claims beyond this are a cruelty to the confiding patient. In functional diseases we have grounds for great confidence in our therapeutic measures, but we should make ourselves sure by a diligent study of the pathology of any condition in question that we are not confusing remission with cure.

CHAPTER II

PERIPHERAL NERVES

ANATOMY. Cranial and spinal nerves are constituted in a similar manner, except that some cranial nerves are either solely motor, or solely sensory in function, most of them being both sensory and motor, while the spinal nerves without exception are mixed nerves. The fibres of the motor part arise from the cells in the anterior horns of the spinal cord, while those of the sensory find their nutritive center in the cells of the ganglia which are located on the pedicles of the vertebrae. The union of the two parts takes place just outside the ganglion. The cranial nerves get their motor fibres from nuclei in the medulla and pons and crus, which are the analogues of the cell-aggregates in the anterior horns of the cord, while the sensory fibres have a common origin in the widely-distributed nucleus of the fifth nerve. In all cases the outer covering of a nerve is formed by a prolongation of the dura and pia of the cord, brain-stem or brain.

HISTOLOGY. A nerve, as we see it, is a mass of mixed tissue designed for the protection and nutrition of the nerve-fibres in their distribution to the structures of the body, and its essential part is the nerve fibre which we first centrally discover as a filament emerging from a nerve-cell. While it is not now considered certain that it originates in that cell, we do know that its existence and functional ability depend upon the integrity of the cell. The nerve-fibre or axis-cylinder is surrounded by a white, semi-fluid substance which is called the medullary sheath, the white substance of Schwann, or myelin. The form and consistency of this white substance is imparted to it by a network of a fine horny

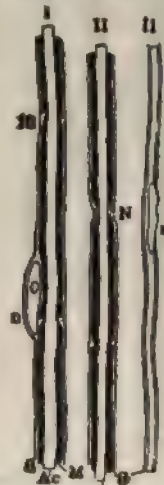


FIGURE 24.—EXHIBITS THE STRUCTURE OF NERVE-FIBRES (FROM GOWERS).

I and *II* show a medullated, and *III* a non-medullated fibre. *Ac*, is the axis-cylinder or neuraxon; *M*, is the envelope of myelin; *S*, is the connective-tissue sheath, or sheath of Schwann; *n*, is the nutritive cell of this sheath; *IS* is the incisure of Schmidt and Lantermann; *N*, is a node of Ranvier. The myelin appears black from the osmic acid stain.

substance called neuro-keratin. Myelin is absent from the structure of the sympathetic or non-medullated nerves. About this myelin is a delicate membrane which is termed the primitive sheath, neurilemma, or sheath of Schwann. Nuclei lie between the myelin and the primitive sheath, one cell to each division of this structure, which divisions are marked off by constrictions by



FIGURE 25.—Shows the degeneration of nerve fibre (from Gowers). At A the protoplasm and nuclei are increased, the myelin is breaking up, the process being more advanced near the nucleus *n*. In B the breaking up has gone on to the formation of globules. In C the latter are much smaller, and many have been removed, so that the fibre is narrow. In D all the products of degeneration have been removed from considerable tracts of the sheath.

which the myelin coating is interrupted. These divisions are called the nodes of Ranvier, and each internodal section, in its growth and degeneration, acts as a unit, and may be considered a single structure with its vital part represented by the cell. In addition there are incomplete divisions, concerned in some way with nutrition, which are called the incisures of Schmidt. The myelin protects the nerve, like the insulating material in an electric cable, and is also nutritive. All spinal nerves contain some sympathetic fibres, but they are absent from the nerves of special sense, except in the case of the olfactory. The nerve-fibres with their coating of myelin are assembled into bundles which are covered by a nucleated envelope of connective-tissue, which is called the endoneurium; larger bundles are composed of numbers of these small fasciculi and covered by another envelope of connective-tissue called the perineurium, and these finally are aggregated into what we know as a nerve by a covering called the epineurium. All these connective-tissue envelopes are continu-

ous, and are ramified by blood-vessels and lymphatics, and the nerves of the nerves (*nervi-nervorum*) which are derived from the nerve-fibres which they supply.

PHYSIOLOGY AND PATHOLOGY. The nutrition of the fibre depends upon the integrity of the cell, and impulses seem to come from it, but not solely, since excitation of fibres can inaugurate impulses. While there are cells in the envelopes there are none

in the fibres, and therefore it is plain that nutrition must come from a cell elsewhere. In addition to nutrition, the cell has some function in the transmission of impulses to and from the brain. Sensory fibres have wide expansions at the periphery, so as to gather stimuli from the widest possible area, but motor cells have their endings in definite and minute primitive particles of tissue. While these nerves in their extra-medullary portion possess a neurilemma sheath and are protected by its vital action, in their intramedullary part, having lost their neurilemma, they immediately undergo degeneration. The first change is a primary degeneration of the myelin; axis-cylinders and nerve-cells are evidently affected later. Degeneration is in the direction of conduction in motor fibres, i.e., toward the periphery, but is contrary in sensory fibres, in their peripheral parts, since their nutrient cells are central to the fibres, but in the cord the rule holds true, since the cell is here peripheral to the fibre. Degeneration is usually secondary, since it generally arises from a primary lesion like the division of a nerve, but there are instances, however, where it is primary, as when disease has its initial site in the nutrient cell. Secondary degeneration is also called Wallerian degeneration. It is not a mere degeneration, but a method of activity of the cell of the node which has apparently been released from some unknown inhibitory influence, and takes on such an exuberant growth that, as in other new growths, fatty degeneration ensues, and destruction of the tissue is a final result. In the rabbit, within twenty-four hours after the division of a nerve, the medullary sheath breaks up into fragments, by the end of the second day these break into smaller fragments, and interruption of nervous conduction is for the first time evident. This process is quite complete by the end of the fourth day, and then these small fragments become converted into fat globules, a process of saponification ensues, and the substance passes out through the neurilemma, and is removed by the lymphatics, so that by the end of the second week the neurilemma is an empty sheath, with the possible exception of some finely granular deposit. In central lesions this process begins simultaneously at several points, but in peripheral nerves it proceeds from the center toward the periphery, except in the rare peri-axillary type of Gombault, seen in poisoning by lead and alcohol, where degeneration takes place from numerous centers. Simple division of a nerve seems to be less serious in its consequences than an extensive dislocation of myelin by a contusion. All severe changes produce degeneration of the end-organs in the muscles, and in the case of motor-nerves produce nutritional changes in the muscles. Regeneration is a slow process, taking from two to four months after a simple division. Nerve-conduction, and the possibility of excitation, do not always progress at the same rate.

NEURITIS.

DEFINITION. This is an inflammation of a nerve or nerve-trunk, or the degeneration of nerve fibres. The resulting symptoms of the interstitial forms are the effects of a compression of the nerve-fibres in the nerve-trunk by an inflammation of the connective-tissue envelopes, rather than of the fibres themselves.

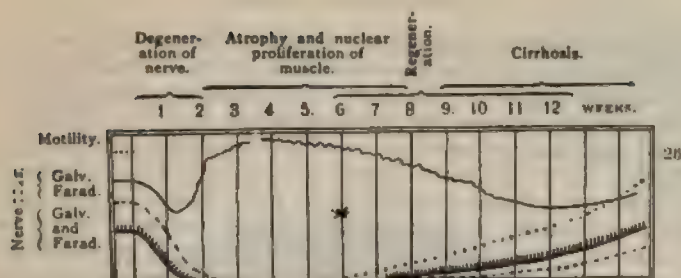
DIFFERENTIAL DIAGNOSIS. Should be differentiated from Neuralgia and Hysteria.

ETIOLOGY. This may arise from traumatism, or from the action of chemical agents, sometimes from cold (perhaps not often directly, but by lowering the powers of resistance to causes of infection which are always present), by infections, chronic intoxications, and the various cachexias.

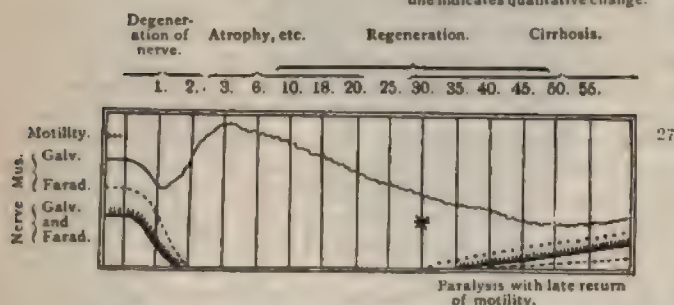
PATHOLOGY. It may be in the perineurium, or be a general interstitial process, or be parenchymatous, i.e., in the nerve-fibre itself, and in all cases the different structures participate to some degree. It may be localized in a small section, or be disseminated over a large area, or affect the whole length of a nerve, or affect many nerves simultaneously, or involve them by a gradual extension, as in multiple neuritis. Constitutional states or chronic infections are apt to produce the last form. A perineuritis, or an interstitial neuritis, is an inflammation while the parenchymatous form is a degeneration. There is little difference in their symptomatology. Where there is an inflammation of a nerve, its envelopes lose their lustre, and there is swelling and redness of the connective tissue. If a section is examined with a microscope we find that the blood-vessels are congested, and there may be signs of small hemorrhages; there is an effusion of white cells, and there is to some extent a proliferation of the connective tissue. This process affects the whole nerve, if long continued. In the interstitial cases the process is the same, but begins in the connective-tissue envelopes of the subdivisions of the nerve, and almost certainly affects to a degree the fibres which are less often and less seriously compromised in the epineural type. This interstitial form is most certain to set up proliferation of the connective tissue, and to produce a swelling of the nerve, which is apt to make such a section of a spindle-shape. The parenchymatous form is the most common, and it may be primary or secondary to the two preceding, or arises from injury to the nerve-trunk. It has been thought that all non-traumatic neuritis arose from primary affections of the nerve-cells, although perhaps so slight as to be undiscoverable with our present means of investigation, but that is not as yet certain.

SYMPTOMATOLOGY. Where a single nerve is involved, the advent may be unmarked by any constitutional symptoms, but where

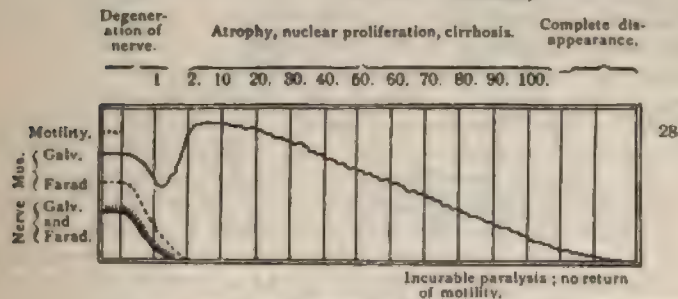
ELECTRO-DIAGNOSIS.



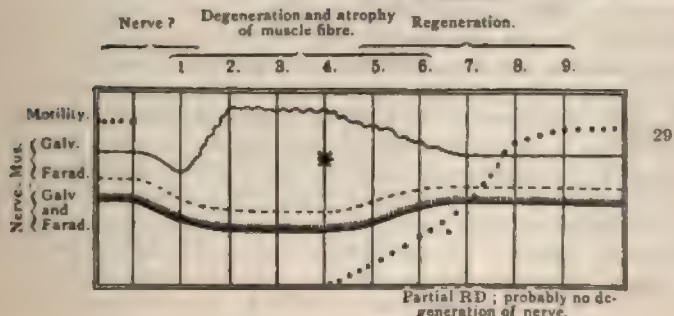
The first ordinate shows occurrence of the lesion and cessation of motility; the star indicates the return of the latter, the waved line indicates qualitative change.



Paralysis with late return of motility.



Incurable paralysis; no return of motility.



Partial RD; probably no degeneration of nerve.

FIGURES 26 27 28, 29. (From O'Connor.)

several nerves are simultaneously attacked there are generally fever and chills, which may be repeated if the process invades new areas with a great degree of violence. The constant symptom is pain, severe, boring, tearing or burning, which may be localized or diffused, and last for weeks or months, but with some variation of intensity. This pain is increased by anything which places pressure upon the inflamed structure, or which puts tension upon it, and it is not merely local, but radiates over the whole distribution of the nerve. A nerve is peculiarly susceptible to pressure at the points where it emerges from a bony canal, or winds about a bone. The swelling may not be conspicuous, but on the other hand it may enlarge the nerve to twice its normal size. The process, it is seen, is not different from that common to all structures of a similar histology, but the results are colored by the fact that structures conveying sensory impressions are involved in it. The symptoms most suggestive are the paresthesias and hyperesthesias in regions remote from the palpable symptoms, but known to be innervated from the nerves in question. This relates to the sensory fibres, but irritation of motor fibres may set up fibrillation, muscular contractions, and an increase of myotatic response. In the first stages tendon-reflexes may be increased, but the violence of the attack may diminish them at once, since the final outcome in all cases is a diminution and final extinction of these reactions. Electrical excitability is increased at first, but it is finally reduced or entirely abolished, so soon as there is a more or less complete break in the continuity of the nerve-fibre. (1st) There is a sharp increase in the response to both faradism and galvanism in the muscle. (2d) After the second day there is a rapid decrease of response to both faradic and galvanic currents up to the tenth day, when it is completely lost. (3d) In the second week, faradism being absent, there is such an increase of response to galvanism that the usual degree of reaction can be invoked by currents of one twentieth the usual strength. (4th) If regeneration takes place, its presence is marked by a decrease of this over-excitability of the muscle to galvanism, and a gradual return of the faradic irritability. The degree of damage to a nerve is determined by the degree of this so-called Reaction of Degeneration. (5th) If regeneration is not accomplished, the galvanic reaction decreases to apparent obliteration for from three to nine weeks, but actually a sluggish response to the anodal (positive) closure may be discovered for a year or more, if the current is very strong. In addition to this change to quantity there is also a qualitative one, in that while normally muscles react more strongly to the negative (cathodal) closure, in the reaction of degeneration the positive is the more potent. The foregoing refers to the reaction of muscles influenced through the nerves at the so-called motor points. When we apply the electrical current to a nerve-trunk, at some

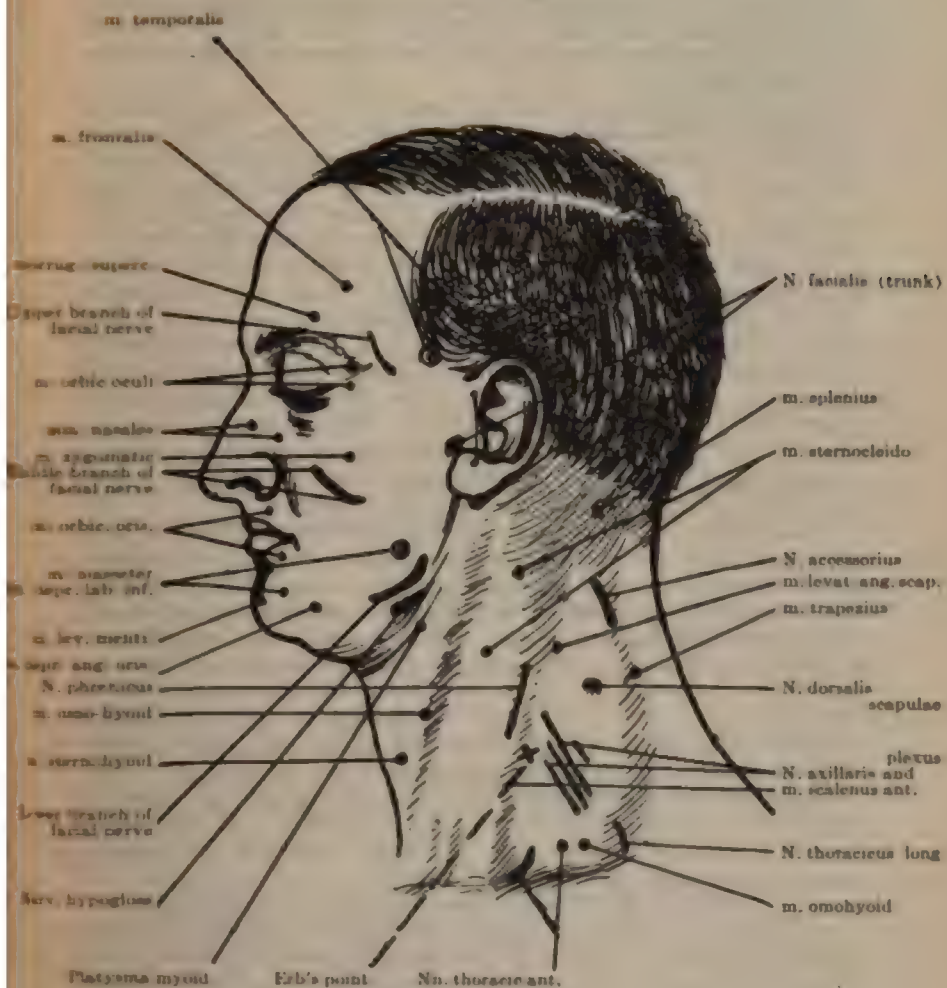


FIGURE 30.—(From King.)

accessible point, its degeneration can be discovered by the fact that it loses its excitability quantitatively only, there being no inversion of the customary response to either pole, and faradism and galvanism both lose their power. There is a slight modal change in the early stages, in that the response is sluggish instead of being prompt.

Trophic disorders, shown by glossy skin, various eruptions, joint-changes, but most of all by atrophy of the muscular masses, are always present to some degree, unless the case is one purely epineural or where the process is so short that recovery antedates such changes. In such cases we know that the fibres have not been involved, or have been only functionally disturbed.

COURSE AND PROGNOSIS. The disease tends to be a chronic one, but may terminate in a few weeks. Some cases endure for years, and not infrequently for six months to one or two years. The cases which gradually develop in a manner suggesting chronicity are apt to last longer than those which are acute from the first, and those from rheumatic, or toxic, or infectious causes have also a relatively good prognosis. The possibility of ascent to the cord from a primary peripheral site is not as common as was formerly held, and is practically true only of those cases where the nerve is the seat of an active infection.

DIAGNOSIS. We base our diagnosis upon the presence of some degree of pain and sensitiveness somewhere along a nerve-trunk, with hyperesthesia and a flaccid paralysis in the distribution of a definite nerve, or group of nerves, corresponding to that of the nerve showing the tenderness. As has been said, temperature may show little or no disturbance, and there is not usually much swelling or redness of the tissues about the nerve-trunk or its distribution. The sensitiveness to pressure is liable to be only slightly developed in the epineural and interstitial types. Parenchymatous or degenerative cases present less symptoms of their true nature than do the other types, since the symptoms are simply those of a break in continuity of the nerve-fibre. In some cases neuritis may be systematized, i.e., affect exclusively the motor, or, in other cases, the sensory fibres only.

DIFFERENTIAL DIAGNOSIS. See Neuralgia, and Hysteria.

TREATMENT. This will be more particularly specified when the separate types are described, but, speaking generally, we may say that it is rest, immobilization, local applications of heat or cold. In the latter stages massage and electricity are of prime importance, and passive movements for the cure of contractures and deformities and atrophies. Nerve-stretching and transplantation, and sometimes resection are also indicated in obstinate cases.

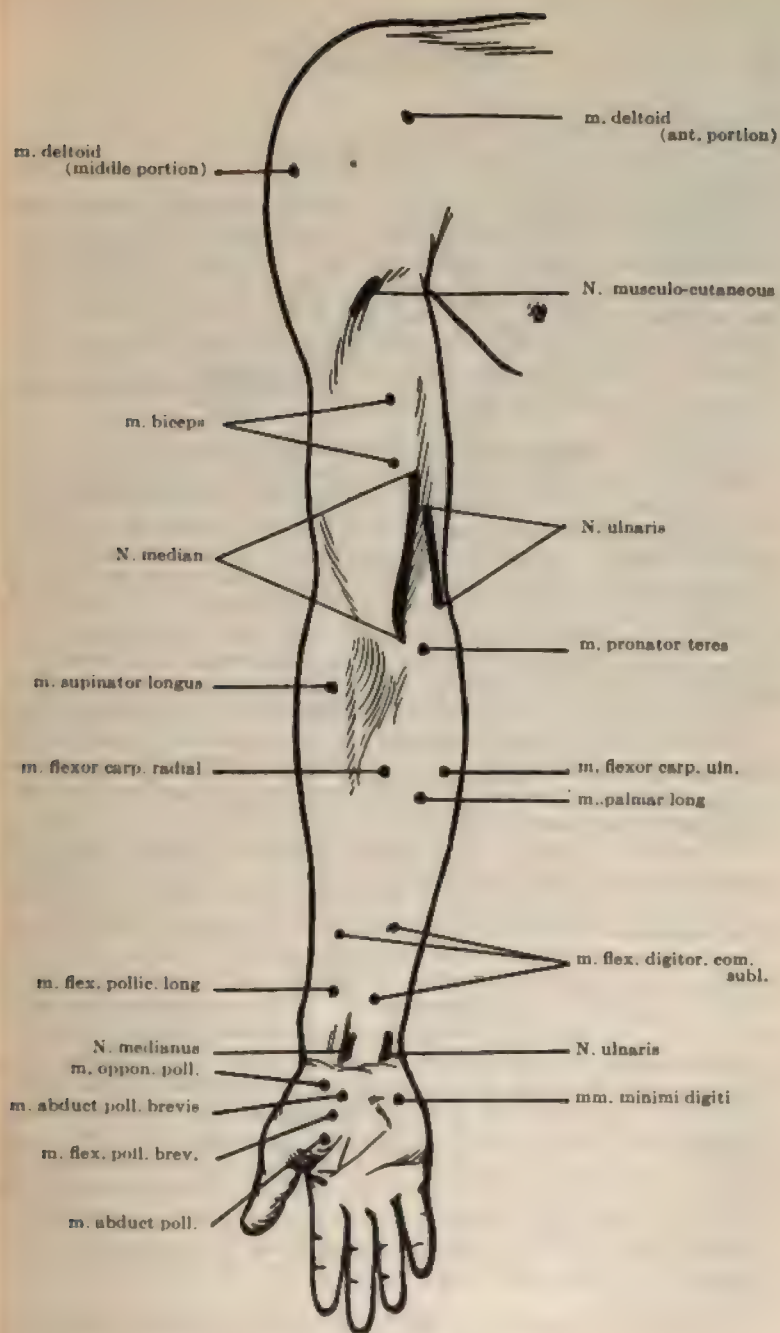


FIGURE 31.—(From King.)

NERVE-INJURIES.

Nerves are frequently exposed to injury, which may range from a slight degree of compression, and of short duration, to the complete rupture of the whole nerve-trunk. An arm is placed in an uncomfortable position for a few moments, and we recognize by the resulting tingling (paresthesia) that the nerve has been compressed, or has been rendered ischemic by an interference with its nutrition. The force was slight, and its duration was short, but its symptoms differ in degree only from those following the most serious injury.

ETIOLOGY. Simple pressure for fifteen seconds, if severe, will break up the myelin, and set up a neuritis of some duration but the nerve-fibre is not susceptible of injury in so short a time. Longer pressure is liable to injure the fibre, but this is not to be precisely gauged by time. All traumata of peripheral nerves act by disintegration of the myelin, or of the fibre itself, thus producing a solution of continuity in the nerve. Fractures or dislocations of bones frequently rupture nerves. They may be caught in callosus, or in new connective-tissue about fractures, or in cicatrices. They may be pressed upon by aneurisms or tumors; it is possible to injure them by the too forcible contraction of a muscle, or they may be caught between bones thrown into unusual, or unusually prolonged, apposition, as in some surgical positions during narcosis, or between the bones of the pelvis and the head of the child in gestation, or, more commonly, in parturition.

PATHOLOGY. Slight compression is often ischemic only, and results in only a temporary functional loss. Longer compression disintegrates the myelin, and seriously affects the nutrition of that section of the fibre for some time. Crushing disintegrates the whole nerve-trunk, while laceration tears it in two, and may separate the fragments to such a distance that reunion is impossible if unaided by surgical measures. If the traumatism has been so severe as to expose the ends of the torn nerve, primary infection may be added. In all, except the last instance, the resulting condition is not an inflammation, but is the Wallerian degeneration described in a previous section of this same article. The resulting degeneration in the central stump does not extend more than one third of an inch, but peripherally it involves the whole length of the nerve, and also its terminals in the muscles and other structures which it innervates. There are possibly no definite trophic cells in the cord, but if the sensory stimuli from the tissues fail to be transmitted to the cells in the anterior part of the cord atrophy always results.

Repair is inaugurated by the proliferation of fine fibrillae from

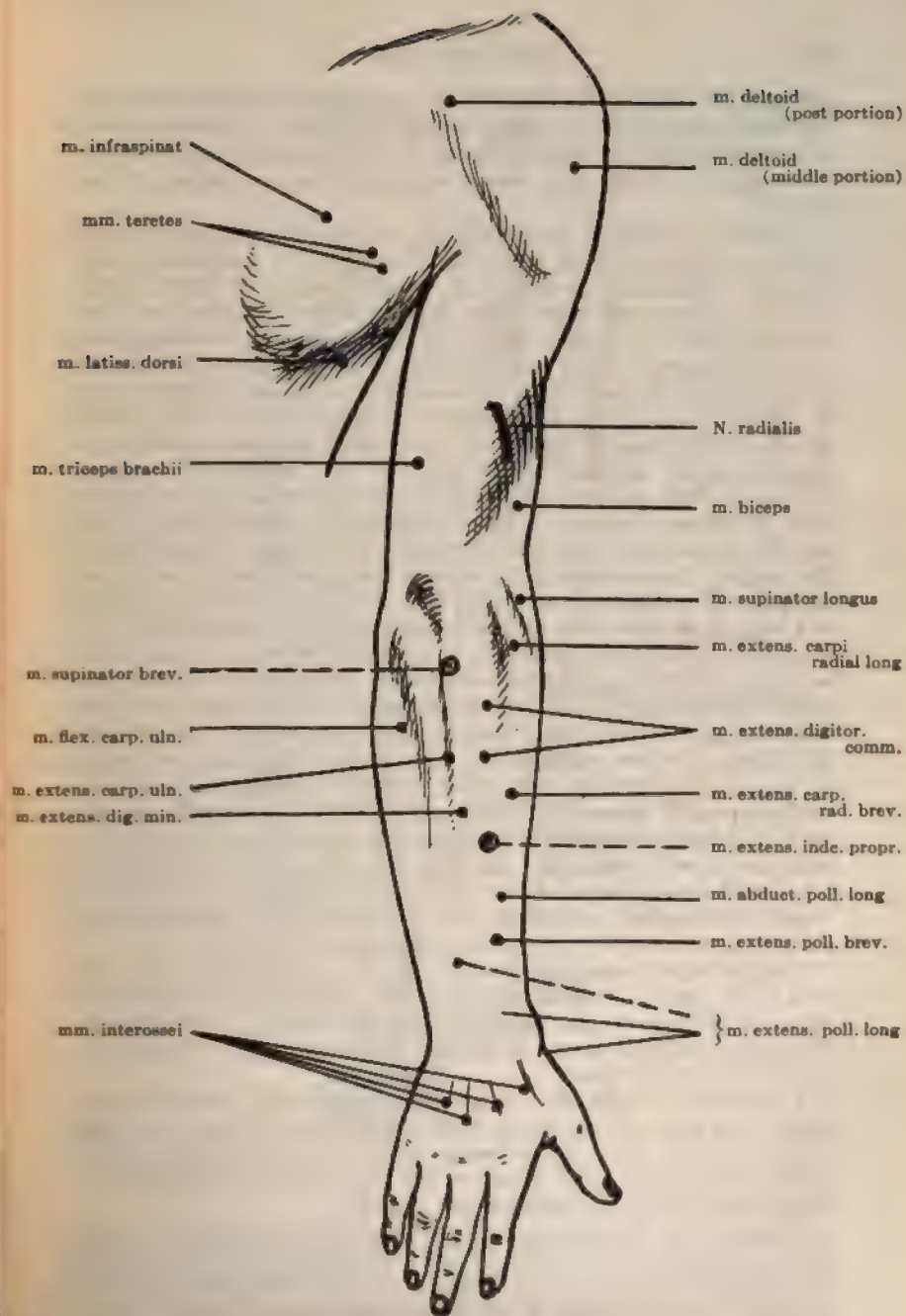


FIGURE 32.—(From King.)

the central stump which seek an anastomosis with the fibres of the peripheral portion. They ramify the new connective-tissue in all directions, and when they finally are re-united, they develop a coating of myelin, and regenerate again into a perfect nerve-trunk.

SYMPTOMATOLOGY. In slight and temporary compression there will be numbness of the parts, and a degree of weakness in the member, which soon passes away spontaneously, or after a little rubbing. In severer cases of the same type the condition may last for some weeks, but, if the force has been sufficient to actually involve the integrity of the nerve, disintegration will ensue in from five to eight days. In such cases the resulting symptoms will differ only in degree in all forms and degrees of injury, except that where the lacerated ends of the nerves have been exposed to the air a greater degree of pain will ensue. In all accidents to mixed nerves we shall be struck by the relatively greater loss of motor than of sensory function. This depends upon two factors. Sensory fibres are more resistant to injury, and owing to anastomosis the sensory innervation of an area is not so dependent upon the integrity of a single nerve. The first symptom of such an injury is a change in sensation in the dependent part (paresthesia). It is at first an increase, followed later by all degrees of diminution and loss. Tingling, burning, numbness followed by diminution of the appreciation of cold and pressure, then of warmth and pain. There is loss of the motor power, since the tonicity of muscles decreases after forty-eight hours, next there is flaccidity, abolition of the tendon-reflexes, and then muscular atrophy. Muscular twitchings and spasm (probably reflex) are not generally prominent, but there is at times fibrillation of muscles. Muscle and joint-sense are rarely disturbed. At first the sensory disturbances are not confined to the area of the nerve actually injured, but are quite widely distributed, yet after the initial shock has passed away these sympathetically disturbed areas become normal.

Trophic symptoms, aside from atrophy, are common, and the skin is affected by simple atrophy, and also by eruptive disturbances of all degrees, corresponding to the gravity of the injury. The joints and nails are also subject to disintegration. Most pronounced in septic cases.

Vaso-motor irregularities develop and the skin becomes blue, there is an increase, or, more rarely, an absence of sweat, and the surface may be cold from ischiemia, or hot from congestion. Electrical changes, and some grade of the reaction of degeneration, or simple diminution of response are found.

COURSE AND PROGNOSIS. Degeneration takes place in from five to eight days. Simple pressure paralysis may recover in a few weeks or even days, but if severe it may be a matter of many months, and if division of the nerve has occurred there will be

regeneration only if the parts are still in relatively close apposition. If a person suffers a relatively slight injury while under the influence of a narcotic, or chronic intoxication, or anesthetic, or the

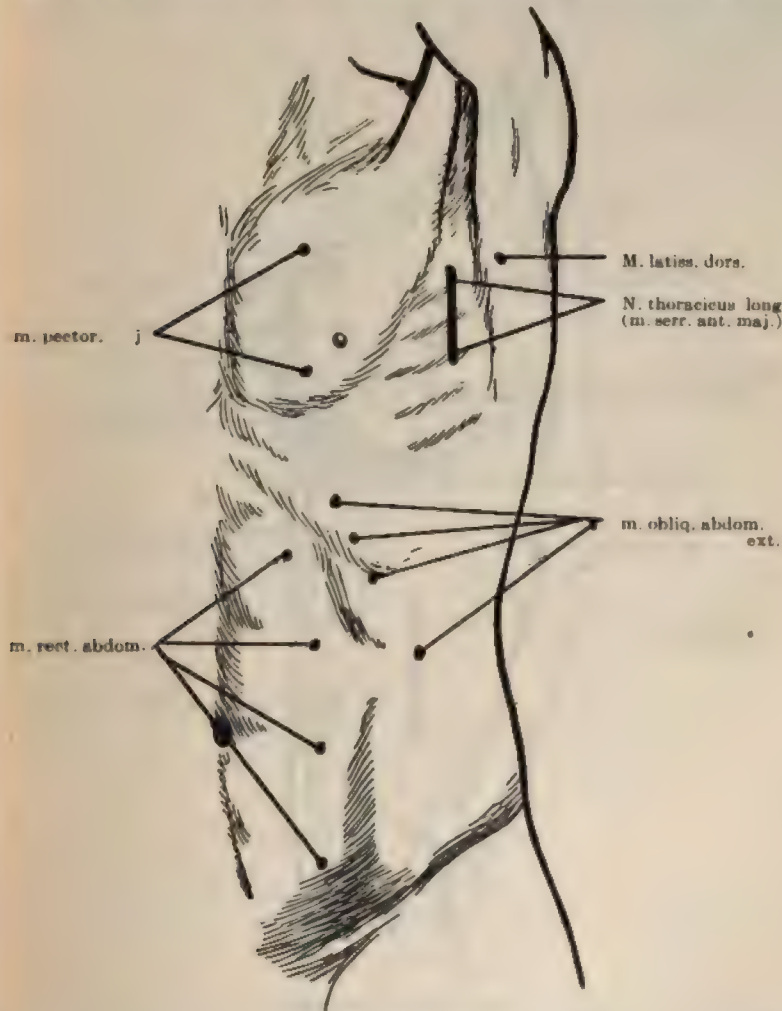


FIGURE 33.—(From King.)

results of an infection, the damage will be much greater, and recovery more doubtful or delayed. Electrical reaction is the best guide. If the reaction of degeneration is present, recovery is

doubtful, and the best that can be hoped is recovery in months; three or four are the least. If after the second week we find a normal response a speedy recovery may be predicted. A partial

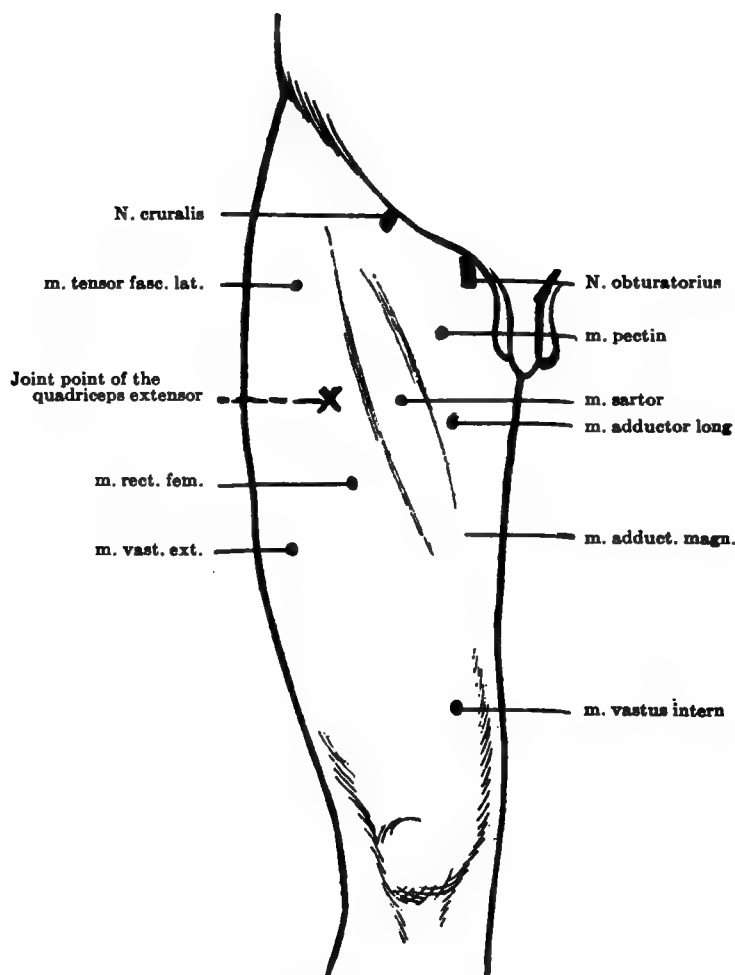


FIGURE 34.—(From King.)

reaction of degeneration gives ground for a relatively good prognosis for recovery, but no time-limit can safely be set. Cases have recovered, even after twenty years, where removal of a physical obstruction to repair was possible.

DIAGNOSIS. In traumatic neuritis the diagnosis lies between that and a paralysis resulting from contusions and injuries to joints and the effects of splints. In joint-contusions, while they may present the same paralysis and muscular atrophies, we find

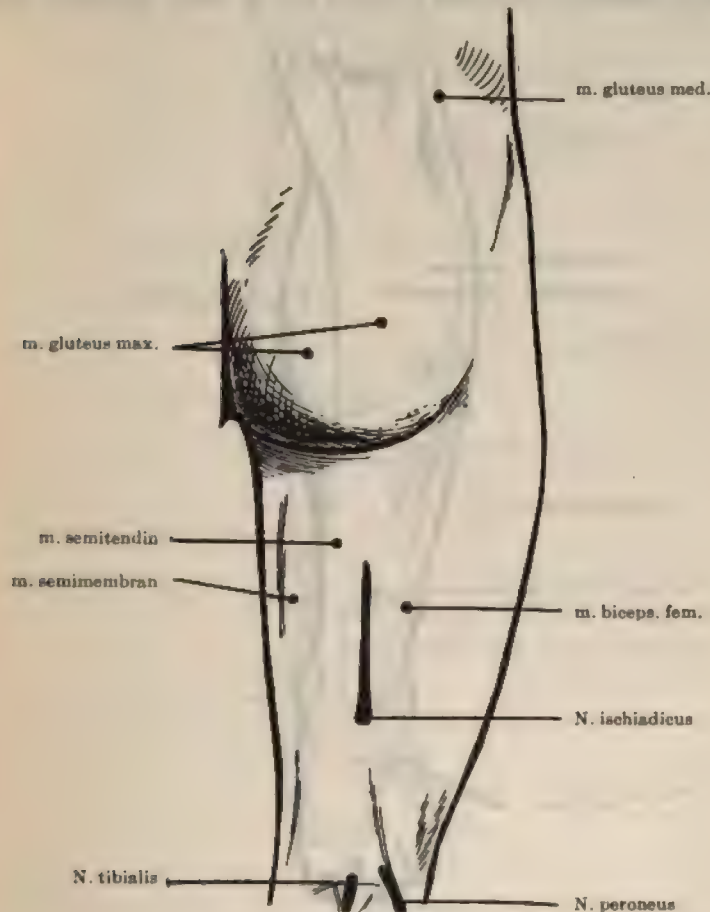


FIGURE 35.—(From King.)

only quantitative changes to electrical stimulation with intact sensibility. In the ischemic muscular contractures which follow the use of splints, we find a history that the hand has swelled, has been painful, and that there have been contractures in flexion in the hand and the fingers. The possibility of movement is lost,

and it is difficult to passively move it, and it causes pain. The diagnostic points are the hardness of the muscles, the stiffness of the joints, the absence of any reaction of degeneration, and the severity of the sensory disturbances. It may be very difficult at times to decide in old cases whether or not contractures are due

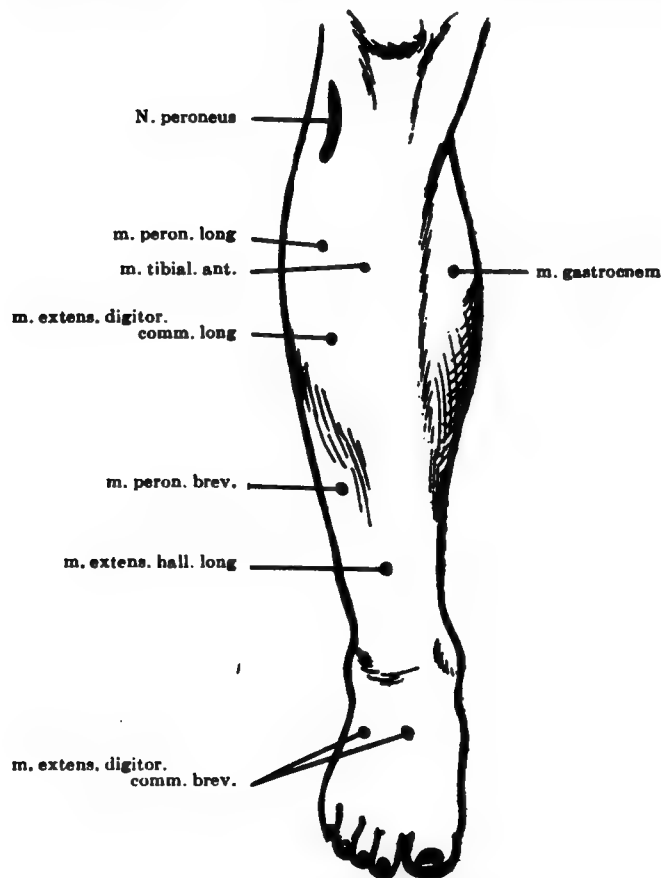


FIGURE 36.—(From King.)

to a neuritis or to peripheral injuries. Here the presence of scars may be the only guide.

TREATMENT. This does not differ in many cases from that of ordinary forms of neuritis, but in others surgery may be the only treatment initially, to be followed by massage and electricity as in the other form. Nerves must be relieved from pressure of

tumors, exudates or callus and intervals between stumps must be bridged by decalcified bone, or strands of gut, or longitudinal nerve-suture must be performed.

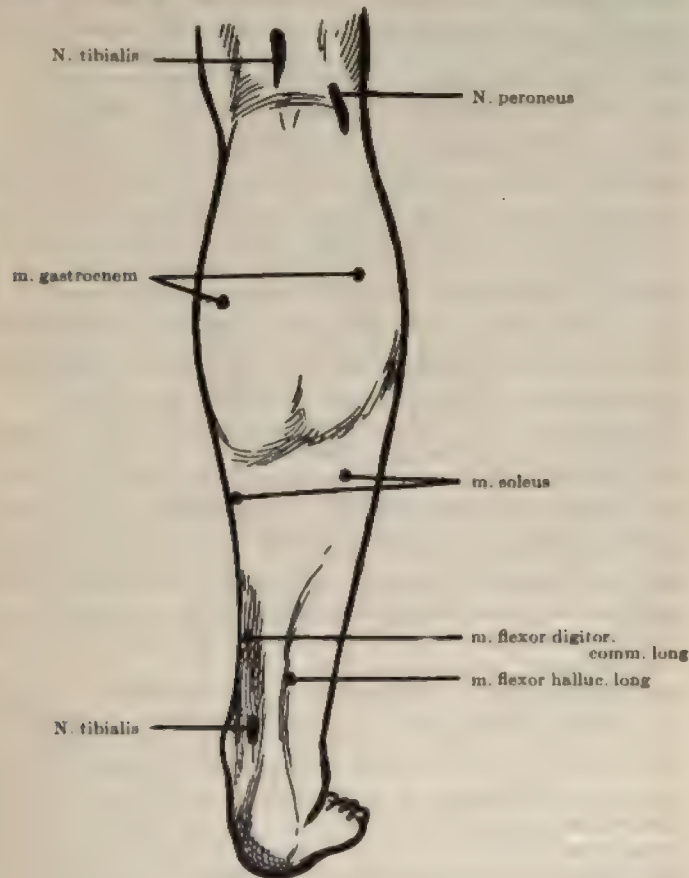


FIGURE 37.—(From King.)

NEURALGIA.

DEFINITION. This is the name given to a severe paroxysmal pain felt in the course or distribution of a nerve or in both localities.

DIFFERENTIAL DIAGNOSIS is from Neuritis and Hysteria.

AGE. It is most common in the active period of life when the organism is most exposed to rigorous conditions and severe strain, and the tendency to it decreases with advancing age, since condi-

tions are apt to be less trying, and the terminals of the sensory nerves and the recipient cells are less susceptible to irritation.

SEX. More females than males are attacked.

ETIOLOGY. The various intoxicants are capable of inducing a neuralgia, and the products of auto-infections, whether they arise from malnutrition, or inanition, or from the cachexias. The toxins of infections produce it very frequently, refrigeration, especially if accompanied by dampness, electrical changes, and sometimes emotional strains. These must all be considered merely exciting causes, since at the root of all attacks of neuralgia we shall find one of two predisposing causes, viz., a neuropathic heredity, or malnutrition. With the same degree of exposure or strain one person will develop a neuralgia, and the other will show no such disturbance, and the determining factor will be that one person comes of a neuropathic ancestry, while the other does not, or the one will be anemic from previous malnutrition, fundamental or acquired, while the immune person will be in vigorous health. The well-nourished person will not escape, however, if his nervous equipment has a large element of inherent instability.

PATHOLOGY. All varieties of degenerative and inflammatory change have been found in the nerves of the neuralgic, but the actual causative lesion is in the cells of origin of the nerve-fibres, or in the cortical representation of this nucleus in the brain. This remote origin is not a theoretical deduction, but is based upon these facts. The resection of a nerve has sometimes been totally useless as a means for arresting the pain, and the nerve has been found to be in a healthy condition. This is a not uncommon result of resection of the fifth nerve for the relief of the pains of tic douloureux, and proves that the lesion is as far back as the ganglion at least. Extirpation of the Gasserian ganglion will relieve some cases which were not relieved by resection of the nerve, which result proves that some cases are actually due to a lesion in the ganglion. Some cases resist this operation, and therefore prove that the cells of the cortex may be the seat of the process. These cases are rare, but evidences that the nerve-trunks are in a normal condition are common enough to give us strong clinical grounds for placing the seat of the lesion in the ganglion cells of the nuclei. Whenever opportunity for investigation has been given cells-changes have been discovered in the nuclei, which are located in the substantia Rolandi in the cord and bulb. This is a gelatinous structure composed of fine nerve-fibres felted together, in which the cells are imbedded.

SYMPTOMATOLOGY. The most obtrusive symptom of an attack of neuralgia is a pain of a burning, darting and shooting character, which is felt running like a stream of fire along the course of a nerve, localizing with peculiar intensity in the area of its distribution. Other nerves in the vicinity are apt to be simultaneously

affected, but in a minor degree, and the skin in this area is apt to be red, hot and shining, particularly if the attack is of long duration. When the pain has subsided the affected area is left exquisitely tender, but the nerve-trunk, which was acutely sensitive to pressure during the pain, is now insensitive as a rule. Pressure upon the nerve increases the pain during the attack, unless it is so great as to temporarily destroy its function, when the result will be a short respite from the pain. Between attacks pressure upon the nerve is often capable of inaugurating a new paroxysm. The knowledge of this possibility teaches the patient to study such attitudes as will preserve the affected nerve from pressure and extension. These attitudes are somewhat diagnostic. If on examination we find that one gluteal fold is smoothed out, and the patient refuses to allow full extension of the straightened leg, we may assume that he is suffering from sciatica, granting that there has been a history of neuralgic pain, while a persistent lateral flexion of the spine suggests intercostal neuralgia. We often find that the effect upon the spinal segments is an irritation of the motor-cells, and there will be spasms of the muscles in the affected parts. The redness and throbbing of the tissues show that the vaso-motor fibres are directly, or reflexly involved in the process, and according to the area implicated there will be sweating, lachrymation, or salivation. The smoothness of the skin is sometimes mechanical from the swelling of the tissues, and sometimes from trophic changes which are most distinctively shown in the crops of herpes which are often present. The hair will fall out over a large area, or in patches which distinctly point out the area of innervation of the affected nerve, or it may only lose its color.

COURSE. The development of a case of neuralgia is usually a matter of only a few hours, but sometimes slight twinges will antedate the actual attack for weeks or months. The duration is an uncertain matter, varying largely with the location. While an attack of neuralgia in the fifth nerve, or one of the brachials, or intercostals may be expected to subside in a few days, an attack of intercostal neuralgia is a matter of a week or two, while sciatica is more likely to harass the patient for the same number of months, and tic douloureux will generally last his lifetime. The individual paroxysms are usually for a few seconds only, but recur every few seconds for hours, and then subside or completely go away for some hours, when another attack comes on. There is often a clock-like periodicity to the attacks. After the initial attack, there are apt to be others for some length of time, especially if there is any exposure or strain. Night is apt to bring some relief, if the part can be kept warm and the position retained. If this is too uncomfortable to be persisted in, there is usually an attack of pain, and then quiet for a period. A person who has

had an attack of neuralgia is extremely liable to a repetition, and the same nerve is almost always the seat of trouble. Neuralgia is rarely bilateral, but may be migratory.

PROGNOSIS. This is always doubtful for actual cure of the condition. Remedies are sometimes to be found which will abbreviate, or terminate the attacks with a fair degree of permanence. A small proportion of attacks of neuralgia occur in persons who have no predisposition, but have developed the condition from severity of exposure, or from the combined effects of exposure and a temporary cachectic condition. In such persons the cure of the attack is the cure of the condition, but this is not generally the situation. It may be said that once a neuralgic, always a neuralgic is the rule. This does not mean that all persons will suffer from a permanent *tic douloureux* who have had an attack of facial neuralgia, or that an attack of sciatica predicates a limping gait through life, but it does mean that that person is very subject upon slight exposure, or strain, or condition of exhaustion to develop a similar attack, so that they may be said to be neuralgic, and have to remember their frailty in all questions relating to the conduct of life. If the patient is young and vigorous, and can give a history of a definite exposure, or some other evident disposing cause, the prognosis is better for immediate relief and future immunity, although we see people of robust development and healthful lives who are chronic sufferers from some form of neuralgia. The fact is, that while they have a good physical development, they are nevertheless inheritors of poorly organized nervous cells.

DIAGNOSIS. A typical case presents an intense, paroxysmal, throbbing pain, initially, and perhaps always, confined to a single nerve and its distribution in the tissues. A later implication of the neighboring nerves does not nullify the diagnosis. The nerve-trunk is either insensitive or sensitive only during the attacks of pain, and for a short time afterwards. The pain is in paroxysms, and there are periods in the twenty-four hours when the parts are free from pain, although after severe spasms they may remain sore to touch. There is generally heat, redness, and signs of tension on the affected tissues. It is never bilateral, but may be migratory.

DIFFERENTIAL DIAGNOSIS. Neuralgia has no certain and universal anatomical basis. It may arise from minute disorders of nutrition in the nerves, or their sheaths, or in the *nervi nervorum*, or in the nuclei of origin in the bulb or cord. Neuritis is due to an interstitial or parenchymatous inflammation of the nerve-trunks. In neuralgia the nerve is insensitive, when pain is absent, or only where it overlies bone, is surrounded by hard fascia, or is superficial. In neuritis the nerve is sensitive to pressure along its whole course, or for a considerable proportion of its extent. The

nerve is not swollen in neuralgia, while it is almost always palpably swollen in neuritis. The pain of neuralgia is diagnostic in having periods of perfect remission. The pain of neuritis is constant, although it may have periods when it is much intensified. Neuralgia is not followed by paralysis or atrophy, except in the late stages of a very severe attack, and in that case even, it is moderate in extent. Neuritis soon produces paralysis and atrophy, and anesthesia of the parts. Neuralgia is apt to be chronic, while neuritis is apt to be acute.

In hysteria the assumed pain is never so bounded that the affection of one nerve, or those to which it is naturally related, could produce it. It is widely distributed, and may involve an entire half of the body. A careful search will detect the presence of some of the stigmata of hysteria, either physical or psychical.

TREATMENT. The acute cases which might be termed accidental in their origin, not depending upon a neuropathic constitution, or a state of general malnutrition, are speedily relieved by proper medication, but those which show a tendency to chronicity or recurrence will demand all the accessory aid of improved hygiene, changes of climate and of occupation. Even with all these aids the results will often fall far below our desires and expectations. In such cases it may be necessary, in order to assure the person any continued immunity from recurrent attacks, to impress upon him that the neuralgia is a proof that his method of life has reduced his nervous system to a condition of irritable anemia, and that functional exhaustion is actually a minor degree of tissue destruction. For such persons the baths, massage, electricity and regular course of life of a sanitarium will often prove a benefit. Massage is always good, but it may prove to be curative only when it is carried to the extent of forcible over-extension of the nerve, as is the method of the Osteopathic practitioners. This has frequently proved efficient in cases of sciatica which have resisted all other methods of treatment. Electricity is often of great value, and the method most approved is the application of the positive pole to the painful area, and the negative on some indifferent point. In some cases a reversal of the customary polarity has been advantageous, and once in a while the faradic current has given better results than the galvanic. The high-frequency current has been highly recommended, and some very good results have followed its use. Vibration has a place in these cases, and the whole area of pain should be covered by the percussion. It should also be applied to the trunk of the nerve. The value of the two last forms of treatment have seemed to the author to be largely suggestive, and instances of an increase of pain from vibration are not rare. It has the least theoretical basis of any of the measures referred to.

In tic douloureux, or in facial pains simulating it, inject a few

drops of a four per cent. solution of cocaine along the course of the nerve for temporary relief, and if the cause of the pain is in the nerve trunk, or its terminals, it will give speedy alleviation, but it will not affect it if it arises from changes in the nucleus. Rhigolene, or chloride of ethyl may be sprayed upon the painful area with advantage, but not for more than three seconds at a time, since the nutrition of the part is already depressed.

THERAPEUTICS. The therapeutics can only be suggested in the space of such a volume as this. Aconite will often relieve pain, when it is like fine needles along the course of a nerve, the attack having been induced by exposure to cold winds. *Actea racemosa* for the pains of rheumatic origin, in those attributed to uterine conditions, in the ciliary region, and when it is intercostal. The pains are sharp and lancinating. Arsenicum for the asthenic and weak patients who show their condition by being querulous and low-spirited, without an adequate amount of pain, and physically it is shown by tremulousness of the limbs. Belladonna for conditions where there is throbbing as a marked characteristic, and the congested condition also affects the cerebral circulation, making the patient irritable and excited. Chamomilla, with the same pains, bears them with as little fortitude as the Arsenic patient, but the physical effect of them is not so great, and the mental attitude is not so much one of despair, as of rebellion, and peevishness. Cedron is valuable in malarial cases, and is clock-like in its appearance. It is stated to be especially adapted to supra-orbital pains on the left side, but has not been so useful in my hands as Cinnabar, which has been almost specific in such cases. Chelidonium, tearing and drawing in the left malar bone, with herpes upon the face and the chin, and the teeth are involved in the pain, and there is evidence of a liver derangement. Colchicum for pains which are left-sided in location, and with a tearing and drawing sensation in the muscles and bones of the face, with cramp-like pain in the maxillary joint. Tingling in the skin of the face as if it had been frozen. Colocynth is a neuralgic remedy, and has been especially recommended for the pains of sciatica, but in this obstinate condition remedies have rarely more than a contributory value. When Causticum is indicated in neuralgia it is generally in cases where the affected parts are also the seat of spasmodic contractions. Gelsemium is to be considered when pains are present in a patient whose muscular relaxation, shown by tremors, would lead one to think of Arsenic, but the mental state has none of the nervous irritability found under that drug. The frequent urination would indicate its use, as in other conditions. Kalmia. Right-sided, burning pain, supra-orbital region mostly affected, and causing ptosis. Magnesia phos. has a general applicability to neuralgic pains, but Nash differentiates between its use and that of Arsenicum, in that those of Arsenicum

are benefitted by the application of heat when they are burning, while those of *Magnesia phos.* are benefitted by the same application when they are not burning. *Mezerium.* A left-sided pain, mostly localized over the malar bone, or eye. *Platina.* A numbness, or tingling, which comes and goes gradually. *Spigelia* for a severe burning, sticking pain upon the left side.

DISEASES OF THE CRANIAL NERVES.

Structurally the cranial are similar to the spinal nerves, except that some of them are purely motor, and some of special sense only, while a part only conform to the spinal type in being both sensory and motor. The 1st, 2d, and 8th are nerves of special sense, and the first is actually a prolongation of the brain. The 3d, 4th, 6th, 7th, and 12th are purely motor, and arise from nuclei which are remnants of the anterior horns which have been cut off by the decussation of the pyramidal tracts. The 9th, 10th, and 11th are mixed nerves. Their motor parts arise from cell-masses lying in the central part of the medulla, and are upward prolongations of the vesicular column of Lockhart Clarke (as it is called lower down in the cord), and are visceral nerves. The sensory parts of these nerves are all grouped into one mass constituting the sensory portion of the 5th nerve. All sensory fibres originate in ganglia which are the analogues of the posterior root-ganglia of the spinal nerves.

Cranial nerves are not subject to functional diseases, for it has been proved that such diseases are localized in the nuclei, or in the brain, since cutting the nerve-trunk, in cases of neuralgia, is not at all certain to relieve the pain formerly experienced in the distribution of that nerve. These nuclei are as liable to infection as are cell-aggregates in other parts. These nerves are means of transmission of impulses only. They, unlike spinal nerves, are very prone to be affected by organic disease. The interstitial structures are susceptible to all the diseases which affect similar structures in other parts of the body, and the fibres are just as liable to be affected by injuries and intoxicants. Their peculiarity of situation rather invites the occurrence of such conditions, and aggravates their effects, since these nerves are distributed to exposed portions of the body, they lie on hard bone in many instances, and they pass through bony canals which become points of painful constriction in the event of a swelling of the nerve-trunk, and the canals themselves are sometimes inflamed. The diagnosis between central and peripheral palsies rests upon the same grounds as in the case of spinal nerves. Central lesions produce weakness of the muscles, with spasticity and increased reflexes, and no electrical changes beyond a degree of diminution

of response to both currents. Peripheral lesions produce a paralysis in place of a weakness, which is flaccid; the reflexes are abolished or greatly diminished, and the muscles atrophy; there is electrically the reaction of degeneration; we find anesthesia or some other variety of sensory change, and nutrition of the parts innervated is impaired.

In the description of the diseases of the various nerves the adjuvant treatment will be separately described under each heading, but the therapeutics will be grouped at the end of the article.

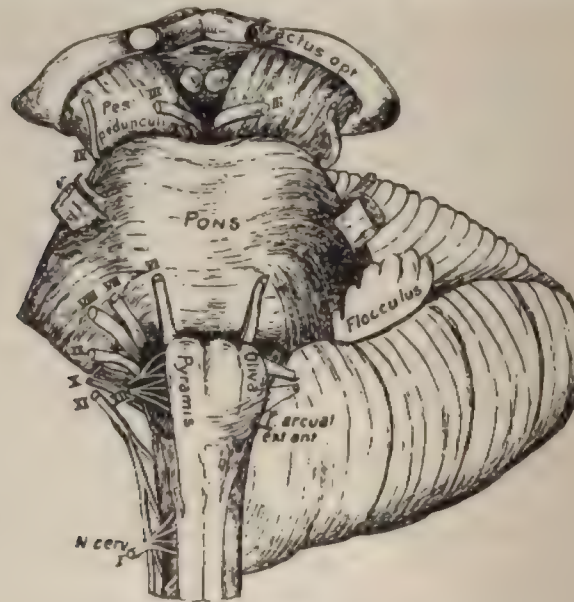


FIGURE 38.—THE BRAIN-STEM.

Crus, Pons and Medulla with a hemi-section of the Cerebellum, showing superficial origin of the Cranial Nerves, which are indicated by the Roman numerals. (O'Connor.)

OLFACTORY (1ST) NERVE. This is actually a forward prolongation of the ventricle of the brain, and, in the embryo, exists as a hollow tube. This rod-like mass runs along the under side of the frontal lobe, and ends in a bulb lying on the cribriform-plate of the ethmoid bone. This plate is perforated by about twenty holes through which drop prolongations of the nerve which are distributed to the Schneiderian membrane, developing bipolar cells as they descend. In the ganglionic bulb is a layer of cells analogous to those found in the cell-nuclei of the cord, while the aggregation of bipolar cells represents the posterior root-ganglion of a spinal-nerve, and the peripheral fibres from these cells are the

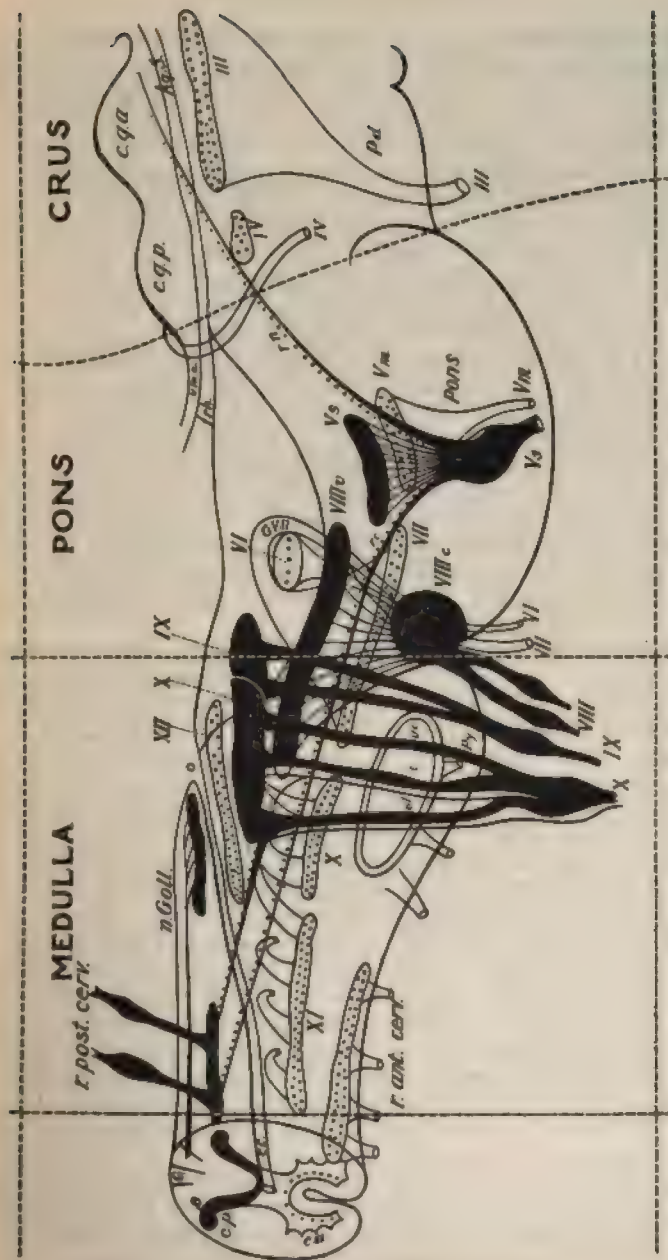


FIGURE 39.—DEEP ORIGIN OF ALL OF THE CRANIAL NERVES EXCEPT THE OLFACTORY AND OPTIC.

Motor nerves, and parts of nerves, are in outline, sensory in black.

analogues of the sensory nerves. The cortical path of these fibres is very complicated, and in some parts in doubt, but it is not incorrect to say that they run first to the uncinate gyrus of the *same* side of the brain, and thence, by way of the anterior commissure, proceed to the optic thalamus of the *opposite* side, and thence to the cortex in the parietal region by a tract in the posterior third of the posterior limb of the internal capsule. Smell is effected by odors acting chemically upon the sensory terminals distributed to the Schneiderian membrane. Loss of smell (Anosmia), diminution of smell (Hyposmia), or perversion (Parosmia), may all be produced by injuries of the anterior cranial fossa, or severe intracranial pressure may produce atrophy of the bulbs, and consequent loss of smell, or tabes dorsalis may cause atrophy of the nuclei. The same result may ensue from the presence of polypi or catarrhal affections of the membrane. As a clinical fact it is well to remember that *perversions* of smell are usually from lesions of the brain, and while found infrequently in degenerations and growths of the cortical nasal centers are more often indications of insanity. In testing a patient for aberrations of smell, one nostril must be closed while the other is being tested, and the mouth also, since many odors are as readily appreciated by the palate. Use alternately the pleasant and the unpleasant, as cologne, and then asafetida.

OPTIC (2D) NERVE. The structure of the optic nerve can best be understood by tracing it from its true, rather than from its apparent origin. At the back of the central part of the eyeball lies a layer of bipolar ganglion-cells like those on the Schneiderian membrane. This layer is the representative here of a ganglion on the posterior root of a spinal nerve. Each bipolar cell sends out a terminal fibre toward the center of the eyeball which is distributed on the retina, by a brush-like terminal, to a connection with the brush-like terminal of either a rod or a cone. In a human retina are 130,000,000 rods, and 3,360,000 cones. This layer of rods and cones has resting upon its outer aspect, i.e., toward the inside of the eyeball, a granular layer of pigment which is chemically sensitive to light. When light is admitted to the eye the granules group themselves about the tips of the rods and cones, and by this alteration in their position give to the observer the sensory stimulus which he has learned to appreciate as light. Toward the brain, or centripetally, run the opposite axons from these bipolar cells in the bundles which are known as the optic nerves. From each eye run from 450,000 to 500,000 fibres divided into centrifugal and centripetal fibres, which may be termed the axis-cylinder processes of the visual cells, while the parts extending into the retina may be classed as dendrites. The great number of these fibres explains why a considerable degree of optic atrophy may exist, and yet the patient be unaware of its presence. The

arrangement of these fibres is in two bundles, an inner and an outer, and the combined bundles from the two eyes cross just in front of the anterior perforated space in what is termed the

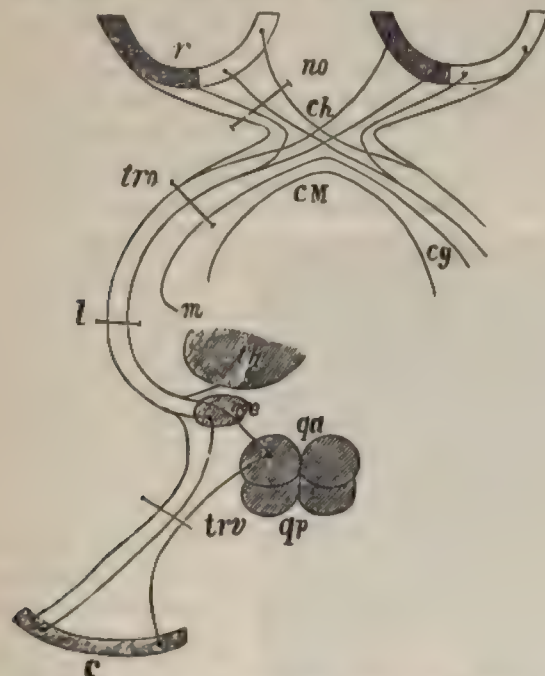


FIGURE 40.—COURSE OF THE OPTIC-FIBRES FROM THE RETINA TO THE CUNEUS.

From Bechterew. The fibres from the inner half of each retina are seen to cross at the chiasm *ch*; the optic tract ends in the external geniculate body *ge*; and the anterior corpus quadrigeminus of the same side *qg*; some fibres may end in the posterior mass of the optic thalamus, *p. th*. New fibres arising from these structures form the optic radiations *tr*, and end in the cuneus *c*. The commissure of Meynert, *CM*, and that of Gudden, *cg*, have nothing to do with carrying light impulses, but connect the large basal ganglia of the two hemispheres, probably. The retina is designated by the part marked *r*. The pupil is not represented, but it is readily understood that rays of light from the right must impinge on the left half of the retina. A lesion at *no* is seen to affect all the fibres from the retina of the left eye; hence no light impulses can pass back, and that eye is totally blind. A lesion in the optic tract, at *tr* or at *l* is seen to affect only the fibres coming from the left half of each retina. Hence each eye is half blind, and in the same-named half of each retina. This hemianopsia takes its qualifying word not from the half of each retina, but from the absent half of the visual field. In the case assumed the lesion is in the *left* optic tract, or in the *left* optic radiations, and there must exist loss of vision in the *right* half of the field—that is, a *right* hemianopsia. A lesion at *ch* will affect the decussating fibres only, and there will result a bitemporal hemianopsia; while to produce a binasal hemianopsia there must be two lesions, one on each side of the chiasm.

chiasm. As they come to the chiasm the outer one, from the temporal half of the eye, continues directly backward and enters the external geniculate body of the same side of the brain, thence to the anterior (called also the superior) corpus quadrigeminus,

thence to the pulvinar of the optic thalamus of the same side of the brain, thence in a compact bundle through the posterior third of the internal capsule, and then, fanning out into what is known as the radiation of Gratiolet, is distributed to the cuneus of the same side, which lies on the inferior surface of the occipital lobe. The inner bundle crosses in the chiasm, but its further course is the same, though it goes to the nuclei of the opposite side of the brain. There is a partial re-decussation of the fibres in the radiation of Gratiolet on their way from the internal capsule to the cunei. From these facts it is evident that complete blindness of one eye alone can only occur when the lesion is in front of the chiasm. These fibres are the centripetal ones, running from the retina to the cortex, and convey sensory impressions of sight. The other bundle is centrifugal and motor, and follows partially a similar route in a reverse direction. The point of divergence is at the anterior corpus quadrigeminus, where a portion leaves the main bundle, and running down the crus in the posterior longitudinal bundle connects with the nuclei of the 3d, 4th, and 6th nerves. There are also probably subordinate connections with other nuclei. This diversion creates a reflex arc by which we reflexly close the eye at the approach of danger to the eyeball. The macular fibres are the seat of the most acute visual perception, and those fibres are in a bundle by themselves, and most protected from injury, and seem most resistant to infection. In the chiasm are crossed fibres from the nasal half of each eye, uncrossed fibres from the nasal half of each eye, uncrossed fibres from the temporal half, and a crossing of the fibres from the two maculae. The fibres to the external geniculate body are from the macula alone.

When the optic nerve is affected, the end showing in the middle of the retina, called the disc, becomes pale and atrophic. Optic atrophy may be primary or secondary. It is primary when the optic atrophy takes place without a preliminary inflammation of the nerve as a whole. There is no choked disc as a preliminary condition from which optic atrophy is to result. It is a primary degeneration of nerve-fibres resulting from disease of their nutrient cells in the nuclei. If secondary, it is almost always bilateral. The primary atrophy is generally from tabes, and is gray in color, disseminated sclerosis (where it is usually bi-temporal and white), general paresis, and, at times, from syphilis. The secondary form after a preceding swelling of the nerve-head, is from intra-cranial tumors (a common cause), renal diseases, lead poisoning, sclerosis, leukemia, pernicious anemia, diabetes mellitus. A very considerable degree of optic atrophy may be present without greatly impairing vision, on account of the resisting power of the macular bundle, but the legitimate result is blindness. Under such conditions we find that the disc is pale, there is concentric contraction of the visual fields and also of the color fields. In this contraction

green goes first. The normal extent of the area in which the different colors can be perceived is white, blue, red, green. Hysteria may produce similar changes and the diagnostic point is this. In hysteria the fundus looks normal, and the loss of vision is most apparent on the side of the body on which the loss of the sense of touch or pain is most evident. The loss of color perception is also eccentric in that the field for red or green may be larger than that for white, or there may be a symmetrical reversal.

Keeping in mind the anatomical points just discussed, it will be evident that the lesion of one optic nerve between the eyeball and the chiasm is the only lesion that will cause complete blindness in one or both eyes, according to the depth to which it penetrates the chiasm. Another reason for the usual escape of the macular bundle is the fact that it lies in the center of the chiasm, and a lesion must be deep to affect it. Central vision will thus escape in all partial lesions. If the external portion of the chiasm is injured, all the fibres going to the temporal half of that eye will be involved, and such a loss will cause blindness to objects approaching the nasal side of that eye. We can get blindness of both temporal halves, seen on the nasal sides, only from a double lesion. This is heteronymous hemianopsia. Nasal hemianopsia has clinically never been found from a lesion behind the chiasm.

If a lesion occurs in the anterior part of the chiasm, the fibres coming from the nasal half of both eyes are affected, and blindness to objects coming from both temporal sides will result. This is heteronymous hemianopsia. If a lesion affects the optic tract after the decussation has taken place, we shall find a blindness of either the right or left halves of both eyes. This is homonymous hemianopsia. This term indicates it is the same half, either left or right, while heteronymous means the left or right of one eye and the opposite of the other. If a lesion involves the decussation, and also projects into one or the other limb, we shall find that there is temporal blindness of the eye opposite to the affected limb, and total blindness of the other eye. To sum up: we may

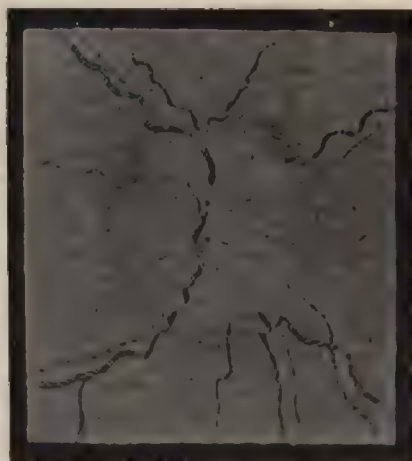


FIGURE 41.—CHOKED DISC.

It will be noted that the clear outline of the disc is replaced by a very indefinite mass of opaque nerve-fibres.

say that from a lesion in front of the chiasm we shall get a blindness of one eye; a partial lesion of the chiasm produces hemianopsia of one or both eyes; complete destruction of the chiasm produces total blindness of both eyes; lesions back of the chiasm produce homonymous hemianopsias; and that in all hemianopsias central vision is undisturbed. The optic atrophy of spinal disease is gray, excepting in the case of multiple sclerosis, while that of primary disease of the nerve is white. The peculiarity in the case of disseminated sclerosis rests upon the fact that it attacks the anterior part of the chiasm. The mechanism of the pupillary reflex demands consideration. There are two sets of nerve-fibres, one contracting, while the other dilates the iris. Light falls

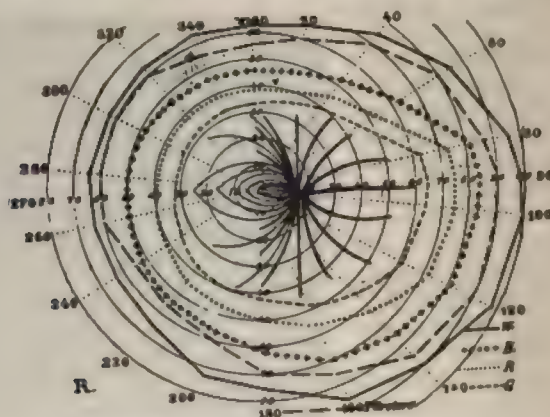


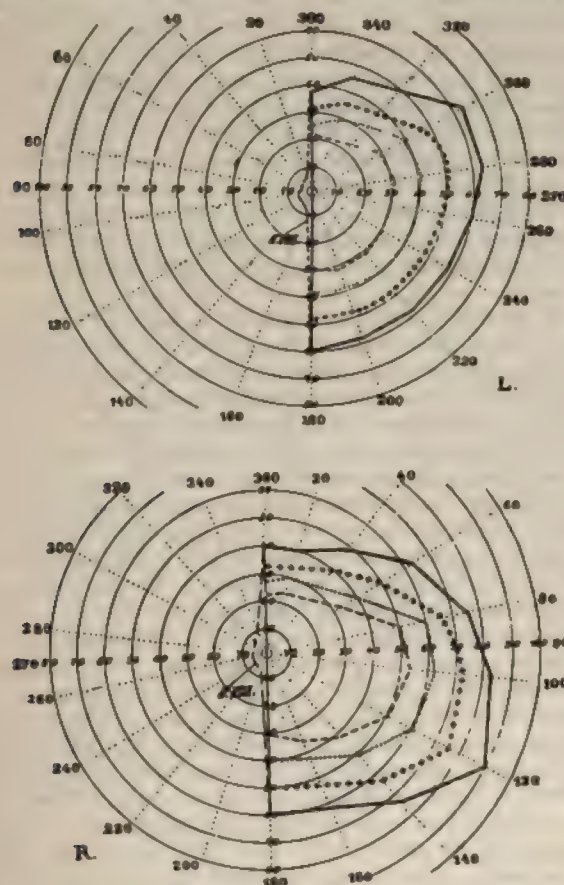
FIGURE 42.—VISUAL FIELD OF THE RIGHT EYE.

The lines indicate the area for white; the stars that for blue; the fine dots that for red, and the large dots that for green. The black lines in the center represent the dispersion of the fibres of the optic nerve, which here have lost their medullary sheath. (From BARR.)

on the retina, stimulates the optic nerve, conveys the impression to the cuneus, whence a motor impulse is sent to the third-nerve nucleus by the path already described. Contraction of the iris is under the control of the third nerve. The pupil therefore contracts. Proof of this lies in the fact that if the optic or the third nerve has previously been divided, no contraction ensues. The third nerve acts through the intervention of the ophthalmic ganglion. The dilatation of the iris is under the control of the sympathetic, the lower center of which is between the sixth cervical and the second dorsal.

OCULO-MOTOR, OR COMBINED 3D, 4TH, AND 6TH NERVES. These three cell-groups, lying under and in the floor of the Aqueduct of Sylvius and of the fourth ventricle, are joined by association fibres into a perfect coördination. The 3d (motor-oculi) nerve

reaches the surface of the inner side of the crus cerebri at the upper surface of the pons, and goes through the outer side of the cavernous sinus, and thence enters the orbit by the sphenoidal fissure. It has upon it the ciliary ganglion (ophthalmic or lenticu-



FIGURES 43, 44.—THE VISUAL FIELDS OF A CASE OF LEFT HOMONYMOUS HEMIANOPIA. The dividing lines between the seeing and non-seeing halves is seen to be perpendicular, but without curving round the centre or fixation point as it usually does. In this instance, however, the field for green is seen to extend beyond the centre. The solid line indicates white, the stars blue, the fine dots red, and the large dots green.

lar), which lies at the back of the orbit between the external rectus and the optic nerve. This nerve supplies the ciliary muscle, sphincter of the iris, levator palpebrae, superior rectus, internal and inferior rectus, and the inferior oblique. Fourth (trochlear)

nerve arises from a small nucleus in the floor of the aqueduct, and is peculiar in that it decussates with its fellow in the roof of the aqueduct just beneath the posterior corpora quadrigemina, and immediately after this makes its exit upon the dorsal surface of the crus, lying upon the superior peduncle of the cerebellum. It then curves about the crus, resting upon the upper border of the pons, passing into the cavernous sinus at its outer side, and enters the orbit by the sphenoidal fissure, and is distributed to the superior oblique. Sixth (patheticus) nerve has its nucleus in the floor of the 4th ventricle, quite a little below that of the 3d, and 4th, the connections of the three making possible the conjoined movements of the eyeball. It emerges just at the lower margin of the pons and near its center, and enters the cavernous sinus at its center, lying just under the internal carotid artery, and enters the orbit by the sphenoidal fissure, and is distributed to the external rectus muscle.

Paralyses of these nerves arise from injuries to them anywhere in their course from their nuclei to their distributions. They may be injured by new-growths, exostoses, especially vulnerable to the effects of thrombosis of the cavernous sinus, chronic inflammatory conditions and tuberculosis, whether in the bones, meninges, or brain-substance. The exudates of syphilis or tuberculous meningitis may permeate their structure, or may compress them in continuity. In the orbit they are open to damage from hemorrhages, thrombosis, or new-growths, and there may be a parenchymatous degeneration from cold or alcoholism, or an interstitial inflammation may be a result. The infections are the most common causes of a degeneration of the fibres, and of these infections diphtheria is the most potent. This infection is the most common cause of a bilateral paralysis of the 6th, although the 3d may also be implicated. Disseminated sclerosis often produces a partial or complete paralysis of the oculo-motor group, and tabes, or general paralysis of the insane (general paresis), are the most frequent causes of pupillary rigidity. Paralysis of the oculo-motors causes giddiness, diplopia, squint and deficient movements of the eye toward the point of origin of the affected nerve. It should be remembered that there is a form of migraine which is marked by a transient oculo-motor palsy, especially of the 3d nerve, which may occur at regular intervals, and be followed by complete recovery. Charcot thought it depended upon an irritation of the vaso-motor fibres. At times it is accompanied by a blunting of sensation in the distribution of the 5th nerve.

TREATMENT. Syphilitic cases should be energetically treated. Other cases should be treated on the basis of their causation. If the eye continues to deviate after treatment use electricity. Apply a bipolar, faradic electrode over the conjunctiva after cocaineization, at the insertion of the muscle. Use the very weak-

est current. Another method is to apply a galvanic current over the eyelid, or along the margins of the nose and the inner margin of the lid, with the negative sponge electrode, and place the positive on the nape of the neck. Hemianopsia is to be treated causatively.

FIFTH (TRIGEMINAL) NERVE. This is a mixed nerve, and the motor-root rises in a nucleus in the pons. The sensory roots are three in number, and actually have their origin in the Gasserian ganglion which lies in the *cavum Meckelii* (a cavity formed in the dura on the upper surface of the petrous portion of the temporal bone). The roots of this run backward to the pons, whence one group ascends to the cells of the aqueduct (formerly termed the descending root), another descends into the *substantia gelatinosa* of the upper segments of the cord (formerly called the ascending root), and arborizes about these cells, and those of the nucleus *cuneatus* and *gracilis* on the anterior surface of the medulla. This mixed nerve appears at the surface of the pons at about its middle, and thence, going forward on the under surface of the tentorium, spreads out as it enters the Gasserian ganglion, while the motor part passes under it. On its emergence from the ganglion the sensory roots are found to have undergone division into three parts, with the lower of which the motor-root unites. These divisions are the Ophthalmic, Superior Maxillary, and Inferior Maxillary. The ophthalmic is about an inch long, and running through the outer wall of the cavernous sinus enters the orbit by the sphenoidal fissure. It is purely sensory, and at the entrance of the orbit gives off branches to the 3d, 4th, and 6th nerves. This branch of the 5th furnishes the sensory innervation to the skin on the front of the head, the conjunctiva, cornea, iris, mucous membrane of the frontal sinus, and a part of that of the nose, and probably sensory fibres to the tear-glands. The superior maxillary branch has a short intra-cranial course in which it passes through the outer side of the wall of the cavernous sinus, emerges from the skull by the foramen rotundum, and enters the orbit by the sphenomaxillary fissure, and passes on to the floor of the orbit. It then, through the infra-orbital canal, passes to its termination in the face. It supplies the skin of the face between the eyes and the mouth, the mucous membrane of the upper jaw, the naso-lachrymal duct, the lower part of the nasal mucous membrane, and that of the gums of the upper jaw, the middle ear, the antrum of Highmore, sensory fibres to the upper teeth, and probably some of the taste fibres.

The inferior maxillary unites with the motor branch, and after a short course leaves the skull by the foramen ovale, and about a quarter of an inch beyond the skull breaks up into its branches. It innervates the skin of the lower jaw, external ear and temporal region of the scalp, the mucous membranes of the tongue, lower

jaw, cheek, lower lip and teeth. It furnishes the motor fibres to the muscles of mastication, i.e., the masseter, temporal, internal and external pterygoids, and to the tensor tympani, stylo-hyoid and the anterior belly of the digastric. Upon the 5th nerve are four small ganglia, each having a motor, sensory and sympathetic root. The ophthalmic, ciliary or lenticular ganglion is 1-12th of an inch in diameter and is situated at the back of the orbit, and imparts sensory and trophic innervation to the eyeball, motion to the ciliary muscle and sphincter of the iris (motor fibres from the 3d), and sympathetic fibres to the dilator of the iris. The sphenopalatine or Meckel's ganglion is 1-5th of an inch in diameter, and lies in the sphenopalatine fossa. Its branches have a very wide distribution to the structures of the face and the sinuses. The submandibular ganglion is of the size of a pin's head, and is situated under the mylo-hyoid muscle and sends branches to the glands under the tongue. The otic ganglion is 1-6th of an inch in diameter, and lies close to the foramen ovale, and sends, most essentially, fibres to the tensor palati and tensor tympani.

Primary disease, excepting neuralgia, is rare. A primary neuritis is unusual and the nerve is rarely involved in a multiple neuritis, but is most often found to be diseased in a secondary way. Any tissue change or new growth in the posterior and middle fossae of the brain, or tumors or softenings in the pons and medulla, will injure the nerve in some of its nuclei of origin, or in continuity. If the softening is in the cord, the so-called ascending root only is compromised, and so the lesion will be wholly sensory, while if the lesion is in the pons, the motor root or nucleus will also be affected. In the cord it has been involved in syringomyelia. Tumors, gummata and exostoses at the base of the skull are apt to involve it, and also growths infringing on the cavernous sinus and the orbit. Operations about the jaw and orbit endanger it in some of its branches. Gummata sometimes affect the nerve directly. If the trunk of the nerve is affected we shall naturally find motor, sensory, trophic, and secretory symptoms, but the sensory losses are not always as great as might be predicted, owing to the richness of anastomosis between sensory nerves in all situations. In such accidents the secretion of tears ceases, smell is diminished, and the sensation in the teeth, cheeks, lips and gums. If we wish to test the sensory condition of the lips we can place a piece of glass between them when the mouth is closed, and the glass will appear to be broken at the point where sensation is lost. It has been said that taste is also lost in these cases, but this is not true, accurately speaking. Taste in the posterior one-third of the tongue is known to be from the 9th nerve, but in the anterior two-thirds from the corda tympani of the facial, but those fibres also actually come from the 9th. The dependence of the preservation of taste upon the integrity of

the 5th nerve is clinically true, but it depends upon the following fact. The production of moisture on the mucous membranes depends upon the sympathetic branches in the 5th, and when that is paralyzed the surfaces are dry, and taste is blunted to abolition, or near to it.

The most evident result of trophic loss is herpes in the distribution of the nerve, and if the ophthalmic branch is affected it will involve the eyeball. We sometimes find partial blindness resulting from lesions of the branches of the 5th, and it is marked by a considerable degree of concentric contraction of the visual field, and some loss of acuteness of vision. It is principally found in diseases of the dental branches, especially those to the molar teeth. If the cerebral centers for the nerve are diseased, we find severe trophic symptoms, such as ulceration of the cheeks and gums, and falling out of the teeth. This is principally caused by tabes. Paralysis of the motor branch involves the muscles of mastication, and, if bilateral, abolishes the reflex. In all peripheral affections there are the reaction of degeneration, and more or less atrophy. In simple compression the symptoms are irritative, and if of the sensory branches it may produce facial spasm, cough, vertigo and other reflex phenomena.

TREATMENT. These paralyses must be treated as are palsies elsewhere, whether they are motor or sensory. If syphilitic they must have specific treatment, and the eyes should be carefully protected. In the severe neuralgic cases, and in *tic douloureux*, the use of injections of perosmic acid must be mentioned for cautionary reasons. Its use has been followed in some instances by blindness, facial atrophy and paralysis. The general result of injections into or near the trunk of the nerve has been disappointing, but recently very favorable reports have been made of the efficiency of the injection of 5-10 minims of alcohol into the nerve as it lies in the foramen.

SEVENTH (FACIAL) NERVE arises from a nucleus in line with that of the 5th and the 9th, and it has a tortuous course in the pons, where it runs backward, and then upward, and then to the outside, and then forward on the outside of the nucleus of the 6th. Before it leaves the pons it is joined by the *pars-intermedia*, which is from the 9th, and embraces the fibres which are afterward to appear as the *chorda-tympani*. It emerges at the base of the pons at the lateral margin of the medulla, and enters the internal auditory meatus, and thence goes to the Fallopian canal, and emerges from the skull at the stylo-mastoid foramen. While in the Fallopian canal it gives off the *chorda-tympani* (taste), and a branch to the stapedius. After leaving the stylo-mastoid foramen it passes into the parotid gland, and on its emergence divides and interlaces its branches to such an extent that that portion is termed the "*pes anserinus*." This nerve which is motor, barring

the pars intermedia, supplies the buccinator (the tensor of the cheek), the superficial muscles of the face, of the scalp, external ear, nose, mouth, eyelids (except the levator palpebrae superioris), and the platysma, the muscles of the tympanum, the levator palati, azygos-uvulae (through the large petrosal nerve), the stylohyoid and the posterior belly of the digastric.

Facial paralysis, prosopoplegia or Bell's palsy, is the most common of all the cranial-nerve palsies, and it is central if previous to the exit of the nerve from the pons, and peripheral at any point outside it. The chief causes seem to be cold and exposure, but probably these are only localizing factors to existing infections of which rheumatism is a common one. The actual pathological change is generally a degeneration of the fibres. Clinically it follows middle-ear disease, caries of the petrous portion of the temporal bone, mastoiditis, tonsillitis, mumps, articular rheumatism, gout, diabetes mellitus, diphtheria, grippe and leukemia. Collier says that conditions primarily affecting the parotid gland are the almost universal cause. Traumatism at almost any point in its course or distribution has caused it, even the extraction of a tooth. A multiple neuritis may extend to the facial. It may be caused by forceps, and is then usually bilateral, and it may arise from atrophy of the nucleus, congenital in time, or from cortical degeneration, or lesion of the path from the cortex.

SYMPTOMATOLOGY. It is apt to come on suddenly in the night without warning, but may be preceded for some days by pain in some of the sensory nerves, in the ear, back of it, in the face or neck, or in the occipital branches. There may be fever, headache, or tinnitus. When we look at the face the lack of symmetry first strikes one's attention, since one side is smoothed out and all expression is gone. The corners of the mouth droop. The patient cannot whistle, since the buccinator is paralyzed, the eye is wide open, the mouth also, and in chewing the food drops from the mouth, or accumulates between the teeth. Taste is absent from the anterior two-thirds of the tongue, although that organ can be protruded naturally. Saliva may be decreased from the affected side, and sometimes there are herpes zoster and swelling of that side of the face. Muscular atrophy is common, there is the reaction of degeneration, and its degree is the basis for prognosis. Hearing is, at times, unusually acute.

DIAGNOSIS. There is no difficulty in well-marked cases, but the localization of the lesion is important. It may be cortical, or in the nucleus, or peripheral. If it is cortical there is no reaction of degeneration, the upper part of the face is spared, and there is apt to be a paralysis of the extremities to some extent. If the lesion is in the pons, just after the fibres have decussated to go to the nuclei, there will be a crossed hemiplegia (face of one side, and extremities of the other), which is a mark common to all pontile

paralyses. Reflexes are either normal or exaggerated. Nuclear paralysis. All branches are usually involved, but the upper face may here also be spared. Reaction of degeneration is present, and the reflexes are abolished. The 6th and the 8th nerves are also involved. If peripheral, we also have the question of a more exact localization, on account of the site at which to apply treatment if any local measures are employed. If it is attacked at the base of the skull, we shall find paralysis of the auditory and other cranial nerves, and the general cerebral symptoms of headache, vertigo and vomiting. If in the nerve-trunk internal to the chorda-tympani, taste and hearing will be affected, while if outside, all the facial muscles will be paralyzed, but these senses will be spared.

PROGNOSIS. In cases from rheumatism the severity will be moderate, from infections doubtful, and if from caries of bone only to be benefitted by surgery. The prognosis is based upon the extent of the reaction of degeneration. If after two weeks the reaction is normal, or only slightly disturbed, the case will recover speedily; it has a good prognosis, though the cure will be long delayed if a partial reaction of degeneration is found, but the prognosis is grave or hopeless if the reaction is complete.

TREATMENT. If the case is syphilitic treat specifically, while if rheumatic hot cloths should be applied to the region of the mastoid, and the parotid gland. Some strongly advocate leeches in the early stages. If it is in the canal, or from caries of bone, the relief must be from surgery. The electrical treatment can be used at all stages. At first stable galvanism, and later labile, and, if neuritis is present, use only the mildest currents. Anode on sternum, and cathode to the nerve where it emerges from the stylo-mastoid foramen. No faradism at first in severe cases, and when it is used, employ very slow interruptions.

EIGHTH (AUDITORY) NERVE. This is a mixed nerve of hearing and equilibrium. It has three nuclei in the floor of the fourth ventricle, and emerges in a groove between the pons and the medulla. It then accompanies the facial into the internal auditory canal, and is divided into two parts, the smaller of which is the upper. This goes to the utricle, and the superior and external semi-circular canals, and subserves the function of equilibration. The lower and larger division goes to the organ of Corti in the cochlea, and sends off twigs to the saccule and the posterior semi-circular canal. This is the nerve of actual hearing. It connects with the pars-intermedia and the geniculate ganglion on the facial. This nerve is rarely the subject of isolated disease, but diseases of the middle ear often extend to its terminal branches in the labyrinth. Meniere's disease is supposed to be caused by a hemorrhage in this nerve. The causes of possible diseases of the cranial nerves are effective in the case of this nerve.

NINTH (GLOSSO-PHARYNGEAL) NERVE. The nucleus is near that of the 10th, in the floor of the fourth ventricle under the fovea inferior. It leaves the medulla at its upper part between the olive and the restiform body, and passes through the jugular foramen along with the 10th and 11th nerves. It is connected with two ganglia, the jugular and the petrous. It runs downward in company with the internal carotid artery and the jugular vein, and crossing the stylo-pharyngeus is distributed to the tongue, pharynx, middle ear and stylo-pharyngeus muscle. It connects with the 5th by the otic ganglion; with the 7th by the trunk and branches; with the 10th in the same way, and with the sympathetic. It is motor to the stylo-pharyngeus, and sensory to taste and touch. It is affected by the same conditions as the other nerves, but is peculiarly liable to suffer from injuries, inflammations, and thrombosis of the jugular vein. Symptoms of its involvement are anesthesia and loss of taste in the posterior third of the tongue and difficulty of swallowing.

TENTH (PNEUMOGASTRIC) NERVE. The roots are in the Trigonum Vagi in the floor of the fourth ventricle. The nucleus ambiguus is supposed to be the origin of its motor root. The sensory fibres are the axons of the root- and trunk-ganglia which lie in, and below, the jugular foramen, and are the petrous and jugular ganglia. These axons enter the medulla and arborize about the cells of the so-called combined nucleus of the 9th and 10th, which is the homologue of the column of Clarke in the lower levels of the cord. The nerve now passes out between the olive and the restiform-body, runs upward to the jugular foramen in which it unites with the accessory portion of the spinal-accessory nerve. It now passes down the neck, and is chiefly distributed to the viscera. It is the longest cranial nerve, since, after going to the pharynx, larynx, heart, lungs, esophagus and stomach, it still further extends to the liver and kidneys. Its functions are motor, sensory, secretory and also vaso-motor.

It suffers like all other cranial nerves, and a primary neuritis is rare. It is peculiarly liable to injury from growths and accidents at the base of the brain, and suffers in multiple neuritis, and from infections and intoxications. In total paralysis from these causes, coincident paralysis of the 9th, 10th and 12th occurs. It is usually implicated in diseases affecting the bulb, in tabes, multiple sclerosis, hysteria, and the neuroses from vascular changes in the jugular vein, and thrombosis of the transverse sinus. In its long course in the chest and abdomen it is liable to pressure and infection from contiguous inflammations.

SYMPTOMATOLOGY. This is very variable with the site of the injury, but if phonation is found deficient we shall find a paralysis of the pharynx, larynx, and fauces. The palate will be paralyzed, and the speech is nasal, it will be difficult for the patient to swallow, and

respiration will be difficult. The pharynx and larynx will usually be anesthetic. If the lesion is paralytic the heart will be accelerated, but if irritative only, it will be retarded. The ineffective respiration will predispose the patient to aspiration-pneumonia, or hypostatic congestion. There may be increased appetite with pain in the stomach, or an absence of hunger. There are thirst and vomiting. Diabetes may supervene. When spasmodic cough, embarrassed respiration and an alteration of voice are present, with or without other symptoms, we should be apprehensive of vagus paralysis. Sometimes in diseases located in the medulla, and at times in hysteria, we find complete aphonia, stridor on inspiration, and dyspnoea, and then we know that the paralysis is bilateral. It has been seen in neuroses following great shock. When it is a neurosis, phonation is more affected than respiration, since respiration is more removed from cerebral control than is phonation. In the neuroses, tachycardia, asthma and partial aphonia have all been observed.

TREATMENT. This must be largely causal. We should antidote syphilis, remove intoxicants, stop alcohol and tobacco, and give a nutritious diet, and good hygienic directions. Massage of the larynx is beneficial in cases showing local symptoms of the parts, and electricity may be of use to stimulate the centers. Tracheotomy may be necessary.

ELEVENTH (SPINAL ACCESSORY) NERVE. As has been said, the accessory part is combined with the vagus, or 10th. The spinal part is the motor nerve to the sterno-cleido-mastoid and the trapezius, of which it especially controls only the upper portion. The accessory portion arises in the same line with the roots of the pneumogastric, and forms four or five small fasciculi which run upward and unite with the pneumogastric in the jugular foramen. These fibres go to the pharynx, larynx, and form an essential part of the recurrent laryngeal nerve, and so are the prominent source of the motor part of the vagus. The spinal portion arises from a long nucleus which extends down the cord as low as the sixth or seventh cervical roots in the lateral column of the cord. They issue from the cord between the ligamentum-denticulatum and the posterior nerve-roots in a number of fasciculi. These form a cord which enters the cranium with the accessory portion. Beneath the jugular foramen it leaves the accessory part, bends backward, usually superficially, to the internal jugular vein, and enters the sternocleido-mastoid muscle which it supplies, and then emerges and enters the trapezius just above the clavicle. Joined here by fibres from the third and fourth cervical, forming the sub-trapezial plexus, supplying that muscle. It is affected by any of the usual causes of nerve-inflammation which occur in the neighborhood of the roots of the cervical spinal nerves. It is subject to primary inflammations which may be either unilateral or bilateral.

A total paralysis of the nerve may only partially paralyze the muscles.

SYMPTOMATOLOGY. The paralysis of the two muscles to which it is distributed, is the most common symptom. Little deformity results, but if unilateral the head cannot be turned to the affected side in a backward direction. It is a form of torticollis, and if both sides are affected the head will fall backward.

TREATMENT is general.

TWELFTH (HYPOGLOSSAL) NERVE. This arises from a long nucleus in a line with those of the 3d, 4th and 6th, extending the whole length of the medulla. It emerges in ten or twelve fasciculi in a groove between the olive and the anterior pyramid. It passes out through the dura in two bundles by way of the anterior condyloid foramen, curves forward under the occipital artery, and reaches up to the under side of the tongue. It is liable to injury from vertebral growths, caries and injuries to the spine, from vascular dilatations and growths in the soft tissues, and diseases in the medulla. On account of the fact that its cortical fibres run to the medulla in close proximity to those to the extremities, it is extremely liable to accident in the case of an apoplexy. Disease in the posterior cranial fossa implicates it, and aneurisms of the vertebral artery.

SYMPTOMATOLOGY. Paralysis of the tongue. A hemi-paralysis is a common incident of hemiplegia; if due to an injury or disease in the medulla it is usually accompanied by the paralysis of other cranial nerves. A hemi-atrophy of the tongue probably never comes from a toxic cause, but it is a common incident in tabes, syringomyelia, multiple sclerosis and the atrophy of other cranial nerves, and it is found in facial hemi-atrophy. Peripheral paralysis of this nerve produces paralysis and atrophy of one side of the tongue, the deviation is toward the paralyzed side, there is incomplete reaction of degeneration, and speech may be only slightly disturbed. It may be slight in complete hemiplegia of the tongue. Recovery is quite possible, since the paralysis is often due to a removable cause.

ELECTRICAL TREATMENT. The method of treatment of these diseases will depend upon the localization of the causative lesion. If it is in the brain, the positive pole must be placed over the motor areas on the cortex for these nerves, while the other pole is to be placed upon the motor points of the muscles. If the lesion has been due to an effusion of blood, or serum, absorption will take place more readily under the influence of the positive pole. If the lesion has been a destructive one, we cannot hope for the regeneration of cells from any form of electrical treatment, but owing to the fact that every muscle is innervated by a very large number of cells, it may be possible that enough of them have escaped damage to give some stimulus to the muscle, and electricity will

develope the nutrition of these cells to its fullest extent. This same rule for polarity should apply to localization of injuries in the nuclei of the nerves in the medulla, or pons. In these cases the positive should be applied to the neck at a point as near the site of origin of the nerves as is possible, while the negative is applied over the motor points of the muscles, or areas of sensory innervation on the skin. Electrical treatment of the motor oculi is of doubtful efficacy, but has some testimony in its favor. The direct method is to apply a mild negative current to each muscle by pressing a knob electrode into the orbit at the point of insertion of the given muscles, the anode at the back of the neck. Another method is to place the cathode over the closed eye, and the anode as before on the back of the neck. Still another method is to place both electrodes over the temples, or in the vicinity of the paralyzed muscles. Except when treating the muscles by electrode in the orbit, pass a current for two or three minutes, and strong enough to contract the facial muscles. In the degenerative conditions of the sclera, which are the result of injuries or diseases of the 5th nerve, the local application of galvanism has a stimulating effect, and it seems to aid in recovery to a very great degree. If in any of these cases, after the injury or disease, the reaction of degeneration is present for more than a month or two, the prospect of recovery of the nerve is very slight. On the other hand, if the nerve speedily responds to faradism, we can feel assured that its recovery is only a matter of time, and when such is the case the faradic will often give fully as good results as the galvanic current.

ANATOMY OF UPPER PLEXUS.

In considering this subject we must rely for a diagnosis of the site of injury upon two factors, viz., the exact location of the motor palsy and of the sensory losses. Some divisions of a spinal nerve are motor only, while others are as exclusively sensory, but, for the most part, they are mixed in function. If we, however, find an area insensitive, which is subserved by a nerve which is exclusively sensory, and at the same time discover a motor palsy in an area whose nervous supply is exclusively motor, we are at once made sure that the lesion is at some point common to both of these nerves. Carried to its full signification this divides our investigations into the two natural divisions of palsies of separate nerves, and of the brachial plexus, as a whole, or in part. In order to make the subject clear in the least possible space it is advisable to first describe plexus paralysees, since, by that method, the anatomy and pathology of the greater will also comprise that of the less.

STRUCTURE OF THE BRACHIAL PLEXUS. The anterior branches of the fifth to the eighth cervical nerves unite with the anterior

branch of the first dorsal to form the brachial plexus. The roots are increased in size as they go downward, and are placed between the points of origin of the scalenus medius and anticus. The scalenus anticus arises from the anterior tubercles of the transverse processes of the third, fourth, fifth and sixth cervical vertebrae, and is inserted into a tubercle on the upper surface of the first rib. The scalenus medius arises from the posterior tubercles of all the cervical vertebrae, and is inserted into the upper surface of the first rib, just posterior to the insertion of the scalenus anticus. The three upper roots are descending in direction, the eighth cervical is horizontal, and the first dorsal is ascending, and they all unite at an acute angle. The plexus lies between the anterior and the middle scaleni, the three upper roots being above, and the two lower being behind the subclavian artery. They are thus exposed to damage by dilatations of this artery. Remember that the seventh cervical segment of the cord lies at the level of the top of the shoulder when the arm is extended. The plexus now descends obliquely through the supra-clavicular fossa, passes under the clavicle and the subclavian muscle, and is covered by the pectoralis minor and major. In this position it reaches the axilla, where it lies between the subscapularis and the serratus anticus. The axillary artery lies below the clavicle, just in front of the median portion of the plexus, and in the axilla it passes through the two divisions of the median nerve, and gains a position to the rear of the plexus. Variations in this position are sometimes found to exist. The plexus divides into its brachial branches in the axilla.

The details in the formation of the plexus are these: The fifth cervical and the sixth unite to form the upper trunk. The seventh cervical alone forms the middle trunk, while the eighth cervical and the first dorsal form the lower trunk, all these unions occurring on the surface of the scalenus medius, and covered by the scalenus anticus. Descending now, and just before reaching the clavicle, these primary trunks throw off an anterior and posterior branch apiece, which combine in the following order to form three so-called cords, named according to their relation to the axillary artery. The anterior branch of the upper trunk, and the like branch of the middle trunk, form the outer cord; the posterior branch of the upper trunk, and the like branch of the middle and of the lower trunks, make up the posterior cord; while the anterior branch of the lower trunk alone furnishes the fibres to the lower cord. At the level of the axilla these cords break up into their final divisions.

The primary branches from the Brachial Plexus are divided into the supra- and infra-clavicular.

SUPRA-CLAVICULAR. Nerve to rhomboids (5 C). Arises from 5 C. nerve before its union with others; nerve to subclavius (5 C).

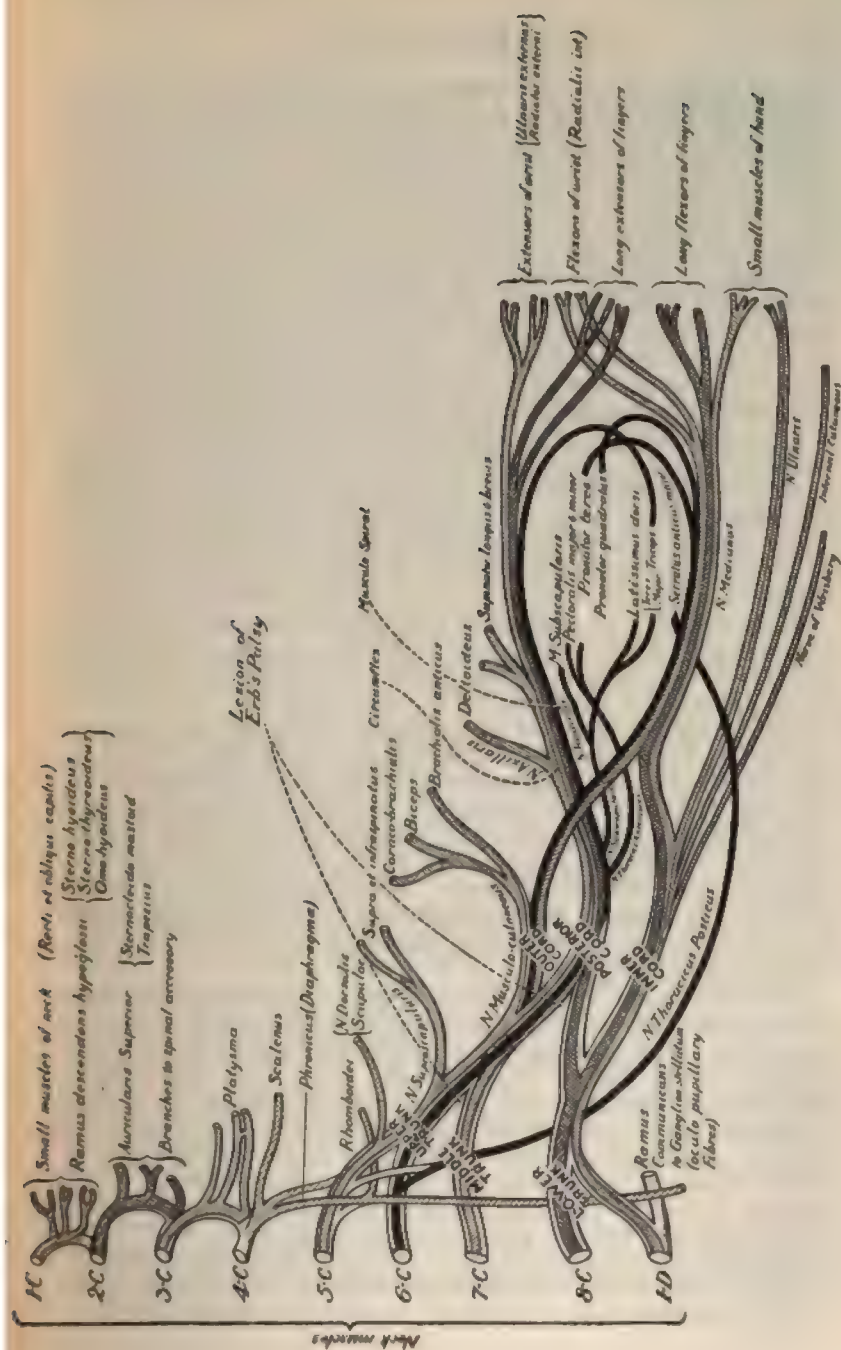


FIGURE 45.—THE BRACHIAL PLEXUS. (Modified from Kocher.)

Supra-scapular (5 C) sends a twig to the shoulder joint, and then divides and supplies the supra- and infra-spinatus. Posterior thoracic (5, 6, 7 C), (external respiratory nerve of Bell). This is made up by the union of the roots from 5, 6, 7 C. The upper two roots give off twigs to the serratus magnus (upper part), and the parts uniting supply the serratus magnus; the three lower cervical supply the scaleni and the longus colli.

INFRA-CLAVICULAR. External anterior thoracic (6, 7 C) goes to the pectoralis major. Internal anterior thoracic (8 C, 1 D) goes to the pectoralis major and minor. Upper subscapular (5, 6 C) goes to the subscapularis. Middle or long subscapular (7 C), goes to the latissimus dorsi. Lower subscapular (5, 6 C) goes to the subscapularis, and principally innervates the teres major.

The outer cord divides into the musculo-cutaneous and the outer root of the median. The musculo-cutaneous (5, 6 C) goes to the coraco-brachialis, biceps, brachialis anticus. After giving off these branches it divides into the anterior cutaneous and posterior cutaneous, both going to the outside of the forearm.

The posterior cord divides into the circumflex and musculo-spiral.

The circumflex (5, 6 C) sends a twig to the shoulder joint, and then divides into two branches. Superior: the upper division is cutaneous; the lower goes to the deltoid. Inferior: the upper is cutaneous; the lower goes to the teres minor.

Musculo-spiral (6, 7, 8 C) branches. Internal cutaneous to the inside of the arm; upper external cutaneous goes to the outer side of the arm; the long external cutaneous to the outer side of the arm, and back of the forearm. It now divides into the radial and posterior interosseous. The radial is the cutaneous branch, and goes to the dorsal surface of the thumb, and the first and radial half of the second finger. Posterior interosseous branch goes to the extensor carpi radialis brevis, extensor ossis metacarpi pollicis, extensor indicis, extensor communis digitorum, extensor minimi digiti.

The Median nerve (6, 7, 8 C, 1 D) goes to pronator radii teres, flexor carpi radialis, flexor sublimis digitorum, flexor profundus digitorum. It then sends out a branch called the anterior interosseous, which supplies the pronatus quadratus; and another branch to the flexor longus pollicis; then is sent off the palmar cutaneous which goes to the palmar surface of the thumb, 1st, 2d and radial half of the 3d finger, palm almost to the wrist. It is the cutaneous supply to, not only the palmar surface of the thumb, of 1st, 2d and radial half of 3d fingers, but also to the dorsal aspect of the last phalanx of all these.

The muscular branches go to the opponens pollicis, abductor pollicis, flexor brevis pollicis, outer half, 1st lumbricalis to 1st finger; 2d lumbricalis to 2d finger; outer half of 3d finger.

Inner cord. This first throws off the internal anterior thoracic to the pectoralis major and minor; nerve of Wrisberg; a cutaneous branch to the olecranon and the lower third of arm—small internal cutaneous (8 C, 1 D). It is divided into the anterior (or external posterior), and internal. The main stem then proceeds as the ulnar.

The Ulnar (8 C) throws off a twig to the elbow joint; a branch to the inner part of the flexor profundis digitorum; a branch to the flexor carpi ulnaris; two inches above the wrist joint it gives off the dorsal cutaneous to the dorsal surface of little, 3d, and ulnar half of 2d fingers; at the middle of forearm gives off the palmar cutaneous to the ulnar half of palm.

The terminal branch of the ulnar supplies the inner surface of the little, and ulnar half of 3d finger; the deep branch supplies inner half of the flexor brevis pollicis, the abductor brevis pollicis, and then terminates in a group of branches which supply the abductor minimi digiti, the opponens minimi digiti, the flexor brevis minimi digiti, 4th dorsal interosseous, 3d palmar interosseous, 4th lumbricalis, 3d dorsal interosseous, 2d palmar interosseous, 3d lumbricalis, 2d dorsal interosseous, 1st palmar interosseous, 1st dorsal interosseous.

NERVE SUPPLY TO SPECIAL MUSCLES:

MUSCLES WHICH MOVE THE ARM. Abductors move the arm away from the body. Deltoid (circumflex, which is a branch of the posterior cord), supraspinatus (suprascapular) from upper trunk (5, 6 C).

Abductors bring the arm to the body. Pectoralis major (internal anterior thoracic, inner cord), (external anterior thoracic, outer cord), coraco-brachialis, (musculo-cutaneous, outer cord); these are also flexors. Abductors which also rotate it inward. Latissimus dorsi (long subscapular, posterior cord), teres major, (lower subscapular, posterior cord); these are also extensors.

Rotators outward. Infra-spinatus (supra-scapular from upper trunk), teres minor (circumflex, posterior cord), subscapularis (upper and lower subscapular, posterior cord).

MUSCLES WHICH MOVE THE WHOLE FOREARM. Flexors: biceps (musculo-cutaneous, outer cord), brachialis anticus (musculo-cutaneous, outer cord, musculo-spiral, a little, posterior cord), brachio-radialis (musculo-spiral, posterior cord).

Extensors: triceps (musculo-spiral, posterior cord), anconeus (musculo-spiral, posterior cord).

MUSCLES MOVING THE OUTER PART OF THE FOREARM. Pronators: pronator radii teres (median, from union of outer and inner cords), pronator quadratus (anterior interosseous branch of the median).

Supinator: supinator brevis (musculo-spiral, posterior cord).

MUSCLES MOVING THE WHOLE HAND. Flexors: flexor carpi radialis (median outer and inner cords), flexor palmaris longus (median outer and inner cords), flexor carpi ulnaris, (ulnar from inner cord).

Extensors: extensor carpi radialis longus (musculo-spiral, posterior cord), extensor carpi radialis brevis (posterior interosseous branch of musculo-spiral), extensor carpi ulnaris (posterior interosseous branch of musculo-spiral).

MUSCLES MOVING THE FINGERS, AND THE FIFTH METACARPAL BONE. Flexors: flexor sublimis digitorum (median formed by the union of the outer and inner cords), flexor profundus digitorum (ulnar, terminal branch of the inner cord), flexor ossis metacarpi minimi digiti (deep branch of the ulnar), flexor brevis minimi digiti (deep branch of the ulnar), lumbricales, outer two (median from union of outer and inner cords), inner two (ulnar, terminal of the inner cord).

Extensors: extensor communis digitorum (musculo-spiral, posterior interosseous branch, posterior cord), extensor minimi digiti (musculo-spiral, posterior interosseous branch, posterior cord), extensor indices (musculo-spiral, posterior interosseous branch, posterior cord).

Abductors: Interossei dorsalis (deep branch ulnar, terminal branch of inner cord), abductor minimi digiti (ulnar, terminal branch of inner cord).

Adductors: Interossei palmaris (deep branch of ulnar which is the terminal branch of the inner cord).

MUSCLES MOVING THE THUMB, AND ITS METACARPAL BONE. Flexors: flexor ossis metacarpi pollicis (median, outer and inner cord), flexor brevis metacarpi pollicis (outer head of median, inner head of median), flexor longus pollicis (anterior interosseous branch of median).

Extensors: extensor ossis metacarpi pollicis (posterior interosseous branch of musculo-spiral), extensor brevis metacarpi pollicis (posterior interosseous branch of musculo-spiral), extensor longus metacarpi pollicis (posterior interosseous branch of musculo-spiral).

Abductor: abductor pollicis (median, from union of outer and inner cords).

Adductor: adductor pollicis (ulnar, terminal of inner cord).

UPPER PLEXUS INJURIES.

Total plexus-paralysis is a possibility, and it may be from disease of the cells of the nuclei of origin, or of the roots, or of the plexus in continuity, and it is often impossible to exactly fix the

site of damage, and rarely is it of importance so to do. Accidents affecting the whole plexus are not uncommon, and are the usual cause of total plexus-paralysis, since a neuritis is very rare with such a localization. Fracture of the humerus or clavicle, or the resulting callus, subcoracoid, or subglenoid, dislocation of the humerus, or hemorrhage, may all produce this form of injury. Such accidents are common in delivery, and sometimes occur in attempting to reduce dislocations. They may be from tumors in the upper clavicular region, or aneurisms of the subclavian. In these accidents, the ulnar most often escapes. After emerging from the plexus, the nerves are always affected singly.

SYMPTOMATOLOGY. After being complete for a short time, it is apt to recede from all but a few nerves. The motor palsy is complete at first, but the sensory is generally incomplete, so soon as the primary shock disappears, and the inner part of the upper arm is apt to escape entirely on account of the vicarious innervation which it receives from the intercosto-humeral nerve. The paralysis is always degenerative.

PROGNOSIS. This is grave, but improvement should not be despaired of for a very long time, as it may slowly improve for many years.

TREATMENT. This is mainly surgical, as the plexus is either ruptured by the violence of the causative injury, compressed by a new-growth, or vascular dilatation, or imprisoned in connective-tissue or callus.

ERB'S PARALYSIS (DUCHENNE'S). This is the most important of the partial paralyses of the plexus, and is of the upper-arm type, as distinguished from the lower-arm type called Klumpke's. This affects the shoulder, and arm in its upper part, on account of an injury to the roots of the 5th and 6th cervical. This may result from forcibly pressing the clavicle against the vertebral column, or the first rib may be the instrument causing the pressure. This condition may be the result of holding the arms upward, outward and backward for a considerable time in surgical positions, and is more liable to produce the injury if the head is, at the same time, turned to the opposite side. The narcosis from anesthetics is also a contributing cause, since the whole organism is, to a degree, in a condition of intoxication similar to that from alcohol, which we know is an essential element in the production of "Saturday Night Palsies," seen in those intoxicated persons who have fallen asleep with the arm hanging down, or flexed under the head. In forcible delivery of a child it is sometimes seen. It may be produced by occupations where heavy burdens are carried on the shoulder, and is here more liable to occur if the person is also affected by some one of the cachexiae, as tuberculosis. It is almost always from injury, but has been the result of primary intoxications; rheuma-

tism, infections, and ptomaine poisoning have caused it. From intoxications it is bilateral, while from injury it is unilateral. It always involves the deltoid (the nerve is the circumflex), biceps (musculo-cutaneous), brachialis internus (musculo-cutaneous), supinator longus (musculo-spiral); often the supinator brevis (musculo-spiral, posterior interosseous branch), occasionally the infra-spinatus (suprascapular), and subscapularis (upper and lower subscapular).

The deltoid abducts and raises the arm, but alone draws the arm forward, while the scapular portion draws the arm back. The biceps flexes the forearm, as does the brachialis internus, while the coraco-brachialis adducts and flexes the arm. Supinator longus turns the forearm to a supine position, while the supinator brevis, which is wrapped about the head of the radius, also supinates the hand. The infraspinatus rotates the arm outward, and the subscapularis rotates it inward. If the supinator brevis is involved, the whole lower arm and hand are in a state of pronation, and cannot be satisfactorily supinated.

SYMPTOMATOLOGY. The paralysis is almost always atrophic, and the reaction of degeneration is present to a variable degree, and the shoulder hangs lower, so that there may be a grade of subglenoid dislocation. When the partially paralyzed muscles are put into action, we may find fibrillation. Pain may be present, but it is never pronounced, though in many cases sensibility is intact, even in the region of the axillary nerves, or it may be very transiently abolished. In severe cases, its distribution is as follows: There is anesthesia in the distribution of the circumflex, and musculo-cutaneous nerves. The outer surface of the upper-arm above the median portion of the deltoid, not quite up to the acromion, and on the outer side of the forearm; there is sensory loss at times in the fibres of the median, going to the thumb and the index finger.

KLUMPKE'S PARALYSIS. A lower-arm type. This arises from an injury of the 8th cervical and 1st dorsal roots. It may be possibly produced by violence in the reduction of a dislocation, but more often arises from some disease of the first rib, or meningitis from syphilis, or operative injuries. Primary neuritis is sometimes a cause, or it may be the final residuum of a total plexus injury. It is a rare form.

It is a disease of the ulnar and median nerves, and as a result, while the arm can be elevated, and the forearm flexed and supinated, on account of the paralysis extension of the forearm is impossible, and the hand is totally paralyzed. Paralysis of the interossei produces an inability to spread out, or to approximate the fingers, or extend the inter-phalangeal joints. Adduction of the thumb becomes impossible, and the paralysis of the flexors of the fingers and the wrist renders the patient incapable of bend-

ing the joints, or of grasping anything in the fingers. The paralysis is atrophic, and the thenar and hypo-thenar eminences are wasted, but there are no vaso-motor symptoms. The sympathetic nerve may be involved, or spared, according as the injury is inside, or beyond the point, where the rami communicantes are given off to the cervical sympathetic from the nerve. If the sympathetic is involved, there is a narrowing of the palpebral fissure, contraction of the pupil, sinking in of the eyeball, and wasting of the face on that side. No dilatation of the pupil results from shading it from the light, or from the instillation of cocaine, and the cilio-spinal reflex (dilatation of the pupil from pinching the skin of the neck) is absent. There are sensory losses in the region of the median, the inner surface of the forearm and upper-arm, and also in the ulnar region of the hand. These may be incomplete.

TREATMENT. Rest of the part, massage, and electricity are the essentials. Rest should be so devised as to avoid strain to the nerves, and when the injury is about the shoulder joint, the arm should be supported in a sling. The extensors of the wrist and fingers should be supported by a splint. Massage of the muscles is always useful. If the muscles will respond to faradism, it may be used, but galvanism is of greater benefit, since it stimulates nutrition more efficiently. Use a current strong enough to produce contractions, 15 to 20 to each muscle, over the motor point, using that pole which gives the best contractions. It is a good plan to give one or two treatments daily. For pain use heat in any form. Keep the part always well wrapped up. Rest in nerve injuries is more important than in paralyses. A mild, constant galvanic current often relieves pain. Use counter-irritation if the pain persists. Mustard and iodine are good, but the actual cautery is the best. Analgesics of the coal-tar series may be needed, while change of scene and climate are often of surprising benefit. Suture is the only remedy for divided nerves. As soon as the diagnosis can be made, it is best to operate. In continued cases it may be necessary to free the nerve from the connective tissue which has engaged it. If end to end approximation cannot be done, attempt to connect by the implantation of animal nerves, or by tubes of decalcified bone. Injuries to sympathetic nerves can rarely be benefitted by treatment. In obstetrical palsy of Erb's type, improvement in position, and some gain in function have followed late operation. The facial type has been helped by connecting into the hypoglossal.

INDIVIDUAL BRACHIAL PALSIES.

Paralyses of individual nerves may be due to disease of the nerve-roots, of the plexus, or of one of the nerve-trunks from the plexus. The only important nerve arising from the roots themselves is the posterior thoracic. The musculo-spiral and the median are most usually the ones affected by the pressure palsies with the added influence of some chronic intoxicant. Secondary forms occur at times, chiefly when the primary neuritis has been from an infection, and the ulnar nerve is the one most commonly primarily affected, out-numbering the other two. Traumatism is the most common cause, but it may also follow toxic and infectious influences, gout, rheumatism, grippe and the intoxications of gestation. Tumors, aneurisms, exostoses may all excite it from pressure. Most common in women, and in persons over forty years of age. While paralysis of the whole plexus is rare, a neuritis of single nerves is common. The reaction of degeneration is present in the muscles from the fact that the integrity of the nerve-endings in them is compromised whenever the nutrition of the nerves is impaired.

Long Thoracic (5, 6, 7 C.) is the nerve to the serratus, and is also called the respiratory nerve of Bell. Its paralysis most often results from injury, or excessive strain from exertion with the arms in an uplifted position. It has occurred after the puerperium from muscle-strain during labor. Some cases seem purely toxic; has been hysterical (possibly). On account of its etiology it usually occurs in muscle-workers, males of the age of twenty-five to forty, and the right side more often than the left. It is not usually involved in upper plexus-paralysis.

SYMPTOMATOLOGY. These are of serratus paralysis. The function of this muscle is to hold the scapula against the thorax, and to rotate it in raising the arm. This paralysis weakens all movements which require fixed positions of the scapula. Inspiratory movements on that side are reduced, but there is no actual paralysis. If from a neuritis there is pain in the neck and shoulder. Sensation is usually disordered. In severe cases there is the reaction of degeneration. The motor points are on the sides of the chest below the axilla.

DIAGNOSIS. This rests upon the fact that at rest the lower angle of the scapula is nearer the spinal column than is normal, and if the arm is raised, the lower angle of the scapula swings out, and is displaced upward by the unopposed action of the levator anguli scapulae and the rhomboids ("Angel-wing Scapula").

PROGNOSIS. It is recovered from in only about one third of the cases. In any case recuperation is tedious, and may take months in a slight case.

TREATMENT. The arm should be supported and the strain should be taken off the scalenius, which means that no movement should be allowed which involves raising the arm. Since the chance of recovery is so doubtful, it has been recommended that the pectoralis major should be split, and a portion of it inserted into the serratus. If local treatment is to be applied it should be over the scalenius, since the nerve is most accessible at this point.

SUPRASCAPULAR (5 C.). This is distributed to the supra- and infraspinatus. Traumatic generally, and may be produced by falls upon the shoulder, and the pressure of heavy burdens. The principal loss is the power of outward rotation of the arm, and some movements requiring great dexterity in the manipulation of the arm as a whole. The paralysis of the supraspinatus simply impairs the action of the deltoid to some extent. It does have some power in rotating the shoulder inward. The only sensory change is a small spot of anesthesia over the scapula. Circumflex usually involved.

SYMPTOMATOLOGY. Rotation of the arm outward is impaired, and somewhat on inward rotation, but the office of these muscles may, to some extent, be assumed by the deltoid and the teres minor. Both these muscles may show a little hypertrophy.

DIAGNOSIS. This rests upon faulty rotation, difficulty in writing sometimes, anesthesia in a small spot over the scapula, and hypertrophy of deltoid and teres minor.

TREATMENT. As above; postural and electrical.

CIRCUMFLEX (5, 6 C.). This supplies the deltoid, teres minor, and goes to the third head of the triceps, and the skin over the lower part of the deltoid. It is usually from falls on the shoulder, or on the hand, from crutch pressure, from continued elevation of the arms in sleep, from rheumatism, and various intoxications and cachexias combined with pressure.

SYMPTOMATOLOGY. The paralysis of the deltoid may not be complete, since the muscle receives some of its supply from one of the thoracic nerves. The deltoid atrophies so that the acromion is exposed. Since the nutrition of the shoulder-joint comes through this nerve, we are liable to find adhesions, and some degree of ankylosis. The sensory symptoms may be slight, but typically there are pain and tenderness in the course of the nerve, and not only anesthesia, but hyperesthesia as well, in the area of its distribution. A sub-glenoid dislocation is usually present.

DIAGNOSIS. This is usually easy from the flattened appearance of the shoulder with the prominence of the point of the acromion, except in a fat person. Here the partial dislocation of the humerus is a guide. The arm cannot be abducted. When ankylosis is a symptom there must be a differentiation between it and a rheumatic or traumatic ankylosis, where a neuritis has been produced. In addition. In these two latter contingencies the scapula will

move when the arm is passively moved, and it will not do so in case of a paralysis. In case of a muscular paralysis from blows upon the muscle there will be no anesthesia, nor reaction of degeneration.

TREATMENT. As above.

MUSCULO-CUTANEOUS (5, 6 C.). This supplies the chief flexors of the elbow, and the skin on the radial side of the forearm, the distribution of the lateral cutaneous. It is rarely paralyzed alone. The brachialis is often spared, since it is supplied by the branches of the musculo-spiral, and the coraco-brachialis is also spared at times, so that a paralysis of the biceps alone has been observed.

SYMPTOMATOLOGY. In general the arm cannot be flexed in a supinated position. It is only possible in a prone position, where the supinator longus is the efficient muscle. There is atrophy on the outer surface of the upper-arm between the insertion of the deltoid and the origin of the supinator longus. There may be some grade of anesthesia on the radial side of the front and the back of the forearm. Reaction of degeneration is present.

PROGNOSIS. Recovery is the rule, and in a moderate case has resulted in three months.

MUSCULO-SPIRAL (6, 7, 8 C.). Supplies the triceps, all the muscles on the back of the forearm, the extensors of the wrist and the fingers, both the supinator longus and brevis, the skin on the radial side of the back of the hand, the thumb, index finger and the radial half of the middle-finger. Most subject to paralysis of any of the individual nerves, on account of the fact that as it winds about the outside of the back of the humerus and the external condyle it is unprotected by any great amount of muscular tissue, and rests upon hard bone. In the upper-arm it is between the triceps and the humerus, and lower down between the brachialis anticus and the supinator longus. Traumatism is almost always the cause, contraction of the triceps in forcible contraction of that muscle has caused it, as in pulling on a boot, crutch pressure, and strains and blows. Infection and intoxication have been doubted, except as accessory to trauma. If the system has been chronically poisoned by any intoxicant or infection, pressure may localize its effects in this exposed nerve, and this is the usual conclusion in regard to "Saturday-night" palsies, or those from narcosis, or the hypodermic administration of various drugs.

SYMPTOMATOLOGY. In crutch palsies the lesion involves the branch to the triceps, but ordinary pressure cases affect the nerve below this point, and the triceps escapes, and the supinators also. Commonly we find a paralysis of the extensors of the elbow and the wrist, the long extensors of the fingers and thumb, and often of the supinators. There is wrist-drop (spastic), and commonly the paralysis is greatest in the index, and decreases regularly in grade

to the little finger (inexplicable). The arm cannot be supinated, and if an object is firmly grasped the contraction twists the arm into a position of pronation. This comes from the paralysis of the supinators, especially the longus. Power of flexion of the forearm is decreased from the paralysis of the supinators. If the paralysis lasts a long time we find Gubler's tumor at the wrist. This is said to be due to either a thickening of the sheaths of the tendons, or to a partial dislocation of one of the bones of the carpus. The loss of sensation is a variable one, and rather uncommon in the arm, while there is a subjective tingling in the hand. If it is a crutch-palsy there is anesthesia chiefly on the front of the arm in the area of distribution of the external and internal cutaneous, but there may be hyperesthesia and anesthetics. In compression cases there is little change in the electrical reactions, but in the severer lesions it is marked. This nerve furnishes fibres to the wrist and carpal-joints, and therefore we may find adhesions and ankylosis.

PROGNOSIS. Generally good, and recovery often ensues in a few days to six weeks without treatment, but if there is complete reaction of degeneration, it cannot be expected for many months. Narcosis-palsy recovers as a rule in from one to four months. The amount of the reaction of degeneration is a useful criterion.

DIAGNOSIS. One fact should be pointed out in considering the diagnosis. This is the most definite instance of the paralysis of a single function, i.e., extension of the hand, and since functions, and not muscles, are represented in particular segments of the cord, we ought to bear in mind the possibility that the losses which we are considering as peripheral may be due to a spinal lesion. Otherwise the peculiarity of the loss makes the diagnosis evident. It is the nerve most commonly affected in multiple neuritis, and therefore the fact must be made certain that it is confined to one arm, and has been so from the first, and that it was sudden in onset. In multiple neuritis from lead also the supinator longus is usually spared; the electrical reactions are more degenerative. Supinator longus paralysis is discovered by placing the forearm midway between pronation and supination. See if movement shows the belly of muscle. In progressive muscular atrophy the paralysis is rarely so complete; it involves both the radial and ulnar, and the electrical reaction, while reduced perhaps, is normal qualitatively for a long time.

TREATMENT. This is mainly postural, and the elbow should never be extended, in order to avoid injury from a contraction of the triceps. The use of electricity is advised after the following manner: Place the positive galvanic electrode on the gluteus, and the cathode on the motor points for the musculo-spiral. Electrodes should be twenty to thirty c.m. square. Increase the current until the patient in endeavoring to extend the hand feels

some ease in the movement. Strength generally needed is six to eight m.a.

MEDIAN (6, 7, 8 C., 1 D.). This nerve goes to the pronators, the radial flexor of the wrist, the flexors of the fingers excepting only the ulnar half of the deep flexor, the muscles which abduct and flex the thumb, and the two radial lumbricales. It collects sensory impressions from the radial side of the palm, on the front of the thumb, of the first two fingers, half of the third finger and, in some persons, from the back of the last phalanx of the index and middle fingers. Sometimes also from the adjacent parts of the ring finger, and the last phalanx of the thumb.

Its cause is usually traumatism received in the patient's occupation. It may come from the long-continued and repeated grasping of an object between the hand and the upper-arm, as necessitated in some trades, from a violent contraction of the pronator radii teres, or from dislocation of the wrist. The fracture of the humerus rarely, of the bones of the forearm occasionally, has produced it. It is sometimes toxic.

SYMPTOMATOLOGY. The symptoms will differ in accordance with the site of the lesion. If injured above the wrist-joint pronation cannot be accomplished completely. After partial action of the flexors, the arm must be separated from the side, and the humerus rotated inward. If the ulnar is preserved we shall find some remaining power of flexion of the wrist and the terminal phalanges of the two inner fingers, and the first phalanges of all. The thumb is in persistent adduction, and cannot be apposed to the fingers, and it assumes a position on the same plane as the fingers ("Ape-hand"). The wrist can be flexed only by a strong inclination to the ulnar side. If the paralysis is peripheral to the wrist-joint the small muscles only of the hand are paralyzed. Pain may be present in the hand. The sensory losses may be absent or conspicuous. There are sometimes atrophy and glossy skin. Electrical excitability and degeneration depend upon the severity of the lesion.

PROGNOSIS. Is generally grave, as the injury is usually a serious one.

DIAGNOSIS. This depends upon the detection of a loss of the power of flexion and pronation, with the distribution of the sensory losses, if present and well-marked.

TREATMENT. As in other forms.

ULNAR (8 C., 1 D.). This nerve supplies the ulnar flexor of the wrist, the ulnar half of the deep flexor of the fingers, the muscles of the little finger, the interossei, some of the lumbricales, the adductor and inner head of the short flexor of the thumb. Its sensory distribution is to the ulnar side of the back and front of the hand, but while it goes to two fingers and a half on the back, it supplies only one and a half on the front. It is superficial

behind the elbow and at the wrist. It is vulnerable to the injuries and intoxications common to other nerves, and may also be paralyzed by continued extension in injurious positions while the subject has been asleep, and has also suffered from forcible flexion of the forearm. Particularly subject to injury in emaciated persons, or those who are cachectic.

SYMPTOMATOLOGY. On account of the paralysis of the *interossei* it renders writing very difficult, the adduction of the thumb is lost, and so is that of the little finger, as well as other movements of that member. The fingers cannot be flexed at the first, or extended at other joints. The weakness is in the little and the ring fingers, and the loss in the others is compensated for by their *lumbricales*. In flexion of the wrist the hand inclines toward the radial side. The hand assumes the "claw-hand" type of deformity, which consists of over-extension of the first phalanges, and flexion of the two distal ones. This is produced by the over-action of the *extensor communis digitorum* and the long flexor of the fingers, because of the paralysis of the *interossei* and *lumbricales* which are their opponents. This is most pronounced in the fourth and fifth fingers, since the *lumbricales* of the other two are still competent from innervation by the median. Complete closure of the hand is impossible on the same grounds. Dupuytren's contraction of the palmar fascia has been a result. Sensory disturbances are variable, and may be very slight, as in cases of complete ulnar palsy the little finger, or even the ball of it, has been the sole area of sensory aberration. Since the dorsal trunk of the nerve turns to the dorsal side at the junction of the middle and lower third of the forearm, and the nerve runs between the ulna and the *flexor carpi ulnaris*, a cutting wound below this point, and on the palmar aspect will not affect the sensibility of the dorsal portion of the hand and fingers. Subjective tingling may be the only symptom. Atrophy hollows out the palm and the base of the little finger, and the thumb may rotate forward slightly. This is not so marked as in progressive muscular atrophy, where all of the *lumbricales* are affected.

PROGNOSIS. Pressure palsies if slight may be recovered from in a few weeks, but severer forms may take months or years, and from injuries a recovery depends upon the possibility of repair. Infection of the plexus takes place more often in injuries of the ulnar than in the case of any other individual nerve.

DIAGNOSIS. This is evident, and the only error which might occur is in considering it peripheral, when a similar palsy is known to occur from a lesion of the cord at the lowest part of the cervical enlargement.

TREATMENT. This is the same as in other palsies of the same locality.

THE LOWER PLEXUS.

ANATOMY. Owing to their position, the common accidents of life do not so readily compromise the nerves of the lower extremity, and, with the exception of sciatica, they do not seem to be so liable to primary forms of neuritis. The other nerves of the plexus are more apt to be involved in a general neuritis, ascending from an infection of one of the nerves lower down. They are liable to injury by diseases of the vertebrae, and of the meninges, abscesses and inflammations of the pelvic viscera, and are peculiarly vulnerable to the pressure and toxic conditions dependent upon gestation and parturition. Paralysis has resulted from section of nerves in operations on the abdomen.

The muscles of the abdomen and legs are innervated from the lumbar and sacral plexuses. The lumbar (L, 1, 2, 3 and half of the 4th) supplies the skin of the lower part of the abdomen, the front and sides of the thigh, and the inner side of the lower leg, and inner side of the foot. It innervates the cremasteric muscle, those which flex and adduct the hip-joint, and those which extend the knee. The nerves to the thigh are the obturator, and the anterior crural. The sacral plexus (lower half of the 4th, L, 5, L, and all the sacral), only the upper four having influence over the leg. This plexus innervates the extensors and rotators of the hip, flexors of the knee, all the muscles which move the foot, the skin of the gluteal region, of the back of the thigh, outer side and back of the lower leg, and most of the foot (all except a strip, on the inner side). These nerves are chiefly branches to the outward rotators of the hip and the gluteal nerve, the small sciatic, and the great sciatic.

SYMPTOMATOLOGY OF LOWER PLEXUS PALSIES. Symptoms of injury to the plexus may be confined to such symptoms as would flow from an injury to the roots. The pain, in a neuritis of the plexus, may extend along the course of the ilio-hypogastric, ilio-inguinal and genito-crural to the lower part of the abdomen, and the groin.

OBTURATOR. This nerve is rarely affected alone, but pressure may be so discrete as to produce it.

SYMPTOMATOLOGY. The thigh cannot be adducted, and so one leg cannot be crossed over the other, and external and internal rotation is weakened. Sensory changes occur in the upper-middle part of the thigh.

ANTERIOR CRURAL. This may be affected either above, or below Poupart's ligament. In the first instance the iliacus is weakened, while it escapes if the lesion is below this point. It may be irritated by a growth near the spine, or paralyzed by it. This nerve is frequently affected in parturition. If the iliacus is

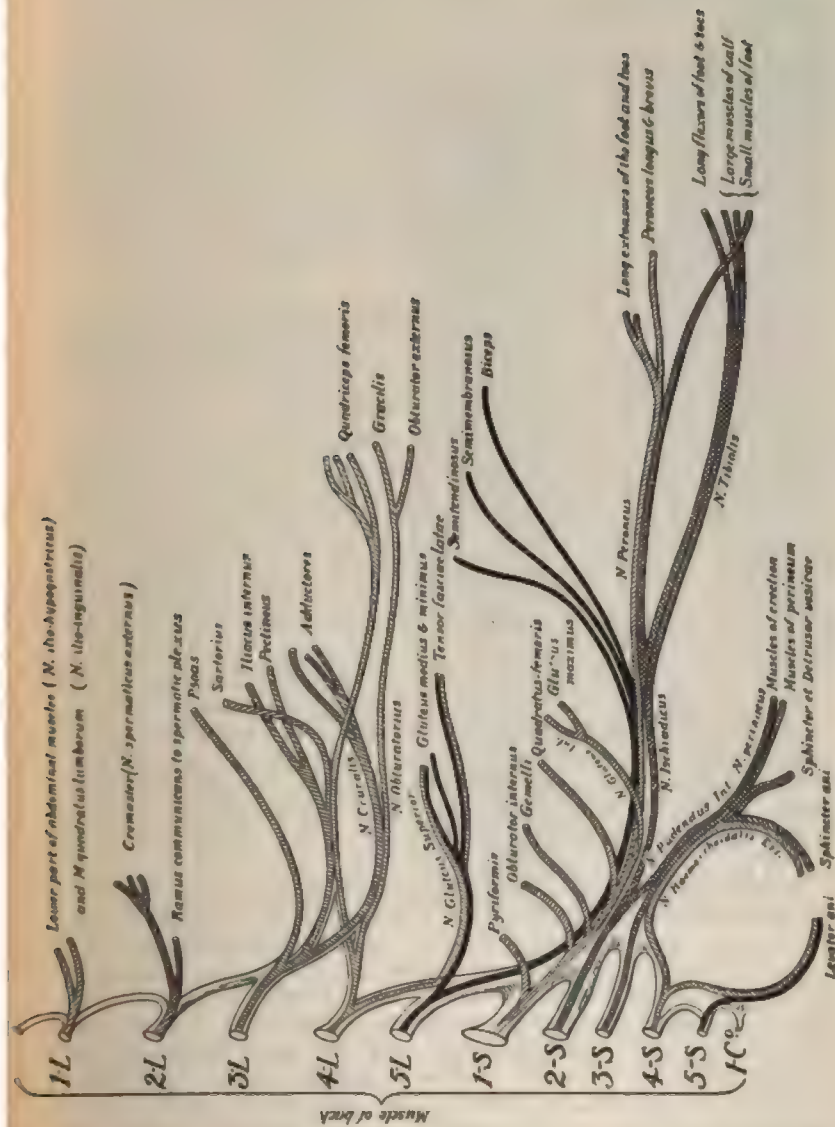


FIGURE 46.- THE LUMBO-SACRAL PLEXUS. (From Kocher.)

involved, flexion of the thigh upon the body is weakened, but if the lesion is irritative only, there is pain in the region of its distribution. In a paralysis below this muscle, the extension of the knee is compromised, and the loss is most conspicuous in walking, since the leg cannot be advanced satisfactorily. Mounting stairs is almost impossible. The usual cause of injury is in fracture of the thigh, and the quadriceps extensor is the muscle almost exclusively paralyzed. Sensory loss or change is from the groin to the inner side of the foot, excepting the buttock and a narrow strip down the back of the thigh, which, below the knee, spreads over the outer side of the calf, and embraces all of the foot, except on the inner margin. Gowers says it also goes to the adjacent sides of the first and second toes.

SUPERIOR GLUTEAL (L, 4-5). Rarely affected in an isolated neuritis, and goes to the gluteus medius and minimus, and the tensor vaginae femoris. It lies between the two plexuses in origin.

SYMPTOMATOLOGY. Its paralysis interferes with abduction, outward rotation, and circumduction of the thigh.

SACRAL PLEXUS. This is rarely affected in a primary way, and sensory diseases practically mean sciatica, from which, secondarily, the plexus may be involved in a neuritis, and in a motor form we are dealing with disturbances of the peroneal nerve. It may, as a whole, be damaged by growths and inflammations in the pelvis, and bruised or compressed in parturition, but the paralysis is due to a combination of an anemic condition and pressure or bruising. Tension from abnormal strains occasionally is the cause.

SCIATIC NERVE (4 L-3 S.). This nerve may be injured from compression, laceration, by growths, or by fractures, and is subject to neuromata. The external popliteal is as exposed as the musculo-spiral, and suffers in like measure from injury, and often is the seat of spontaneous neuritis. It may also be injured by any fracture of the bones. It has been considered subject to neuralgia, but investigation will generally reveal changes in the nerve.

SYMPTOMATOLOGY OF PARALYSIS OF THE WHOLE NERVE. If the injury is near the sciatic notch, there is a paralysis of the flexors of the hip, and of all the muscles below the knee, but walking is still possible, from the possibility of raising the foot by over-flexion of the hip. The leg is thrown forward as a whole, the knee being kept in extension by the anterior crural. The result is a peg-leg, such as we get in hemiplegia, the only difference being that in hemiplegia the leg is spastic. Often the lesion is below the middle of the thigh, and then the flexors of the leg escape. It is not so disabling as a lesion of the anterior crural, the loss of power is usually partial, and the knee-jerk is preserved. Sensory losses from a lesion below the origin of the small sciatic, are in the outer

half of the leg, the greater part of the dorsum of the foot, and all the sole, but it is possible that the leg may escape, owing to anastomosis with other sensory nerves. Atrophy is marked in the back of the thigh and below the knee. It is possible that there may be perforating ulcer of the foot, especially of the ball of the great toe.

PARALYSIS OF THE BRANCHES.

EXTERNAL POPLITEAL (PERONEAL). This nerve is especially liable to injury in the popliteal space, and as it turns about the outside of the fibula. It is sometimes injured in jumping (on hard ground especially), and from taking a false step. Its injury paralyzes the tibialis anticus, the long extensor of the toes, the peronei and extensor brevis digitorum.

SYMPTOMATOLOGY. There is loss of the power of flexion of the ankle, and of extending the first phalanx of the toes. This is objectively striking, in the production of foot-drop and toe-drop, and from the fact that the toes drag upon the ground in walking. From the contraction of the unopposed interossei, the toes curl in upon the sole of the foot, and still further embarrass the sufferer. Contraction of the tendo-Achillis is a common result, and causes the foot to assume the equino-varus position. The paralysis is a flaccid one, and there is the reaction of degeneration. Sensory loss is an anesthesia of the outer half of the front of the leg, and of the

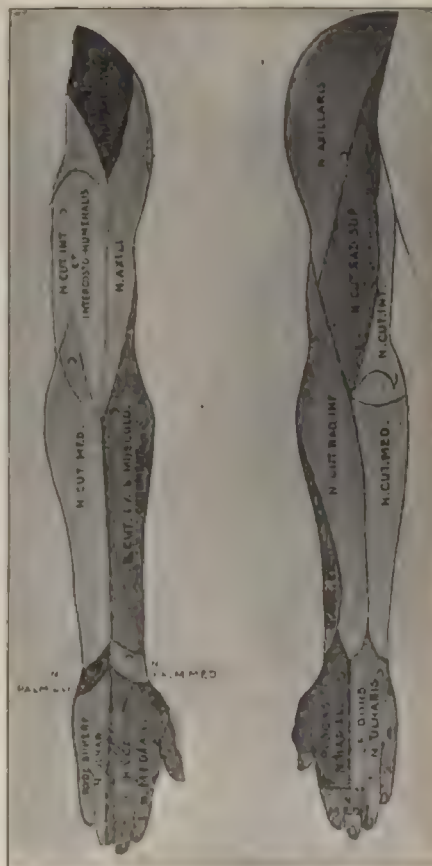


FIGURE 47.—(From Hesse.)

CUTANEOUS DISTRIBUTION OF THE SPINAL NERVES.
UPPER EXTREMITY.

The small curves mark the points where the branches of the nerves pass through the fascia. N. axillaris = circumflex.

dorsum of the foot. The fullness of the muscles on the anterior and outer aspect of the leg is lost.

INTERNAL POPLITEAL. This is analogous to the median and

ulnar in the upper limb, while the external popliteal resembles the musculo-spiral in the course and symptoms of its injury. It is distributed to the popliteus, the calf muscles, tibialis posticus, the long flexors of the toes, as well as to the muscles of the sole. It divides into the internal and external plantar, which may be separately diseased.

SYMPTOMATOLOGY. There is a loss of the power of inward rotation of the flexed leg, if the site of the disease is high enough to involve the branch to the popliteus, and there is a loss of the power of extending the ankle-joint. The result is talipes calcaneus. Sensory loss is variable in amount, but typically it is on the outer part of the back of the leg, and on the sole.

INTERNAL PLANTAR. Paralyzes the

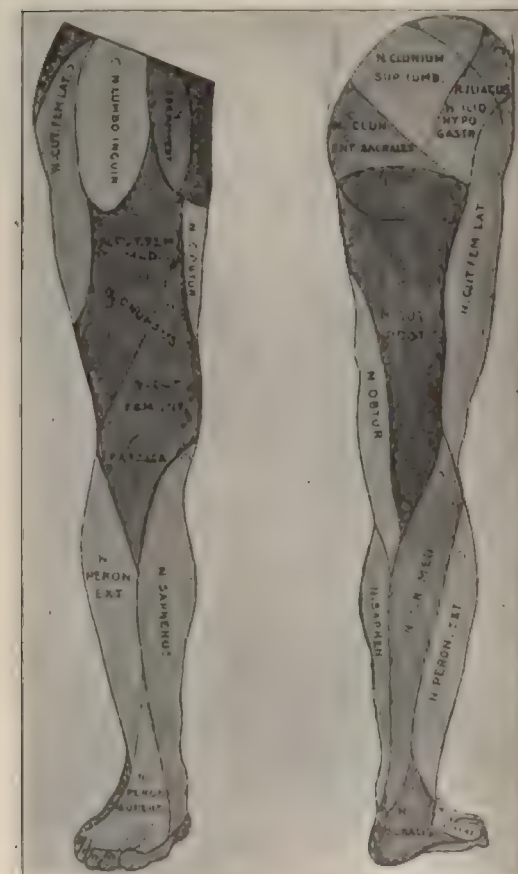


FIGURE 48.—(From Hesse.)

CUTANEOUS DISTRIBUTION OF THE SPINAL NERVES.
LOWER EXTREMITY.

The small curves mark the points where the branches of the nerves pass through the fascia. N. clunium sup. lumb. = gluteal distribution from lumbar nerves; N. clun. int. sacrales = gluteal distribution from sacral nerves.

short flexor of the toes, the plantar muscles of the great toe (except the adductor), and the two inner lumbricales.

SYMPTOMATOLOGY. The toes cannot be flexed at the second

phalanx, and so we find "hammer-toe," and clumsiness in walking. Sensory loss is anesthesia of the inner part of the sole, and the plantar surface of the three inner, and half of the fourth, toes.

EXTERNAL PLANTAR. This is distributed to the flexor accessorius, the muscles of the little toe and all the interossii, the two outer lumbricales, and the adductor of the great toe.

SYMPTOMATOLOGY. This paralysis flexes the last two joints of the toes, and extends the others, from the contraction of the opponents of the interossii. This position of the toes makes walking almost impossible. Sensory loss is an anesthesia of the outer half of the sole, the little toe, and half of the fourth toe.

Metatarsalgia (Morton's painful toe) results from a pressure neuralgia, or neuritis of one of the digital branches of the plantar nerve. It is compressed between the heads of the metatarsal bones, probably as a result of a congenital malposition of the nerve.

TREATMENT of all these conditions is postural, electrical and symptomatic.

LESIONS OF THE CAUDA EQUINA.

Since the cord ends in a conical point, termed the *conus terminalis* at the level of the second lumbar vertebra the fibres emerging at the lower levels of the spine must pursue a somewhat extended intra-spinal course. They take their rise from the lower portion of the cord in close proximity to one another and fall like a coarse fringe which is gathered into a loose bundle by the membranes (*theca*) which extend as a mantle as far as the second sacral vertebra where it resolves itself into a cord (*filum terminale*) which is attached to the fifth sacral vertebra or the upper surface of the coccyx, and so anchors the cord at its lower extremity. The different spinal roots penetrate the *theca* when they come to a point in their descent opposite to their respective foraminae between the vertebrae.

From these anatomical facts it follows that cauda equina disease will be marked by no segmental symptoms, but by motor root symptoms if the anterior fibres are affected, and by sensory root-symptoms if the posterior roots are affected.

ETIOLOGY. Primary disease is rare, since all lesions are generally secondary to disease of the spinal meninges, or to disease or injury of the lower vertebrae or sacrum. Fracture of the spine below the first lumbar is a common cause, bullet-wounds, and hemorrhages from injuries, too slight to actually cause fracture. The meninges are liable to infection in this locality from syphilis, carcinoma, and tubercle, and the latter may produce the damage by the formation of abscess. The lesions are usually partial.

SYMPTOMATOLOGY. In partial lesions the inner fibres, i.e., those

which are sacral and coccyxygeal are affected, and the outer ones only in more complete lesions. If the lesion is a complete one there are paralysis and atrophy of all the muscles of the lower extremities, anesthesia of the buttocks and perineum, and genitals. The knee-jerk and Achilles-jerk are lost, the plantar and anal reflexes also; there are paralysis of the bladder and rectum, and a loss of sexual power. The first lumbar generally escapes, and in this case the sensation is normal in the inguinal region, and the anterior part of each thigh. If the first, second, and third lumbar escape, the anterior crural and obturator nerves retain their function, and only the glutei and the ham-string muscles are paralyzed above the knee, but all below the knee are of course affected. The anus and bladder are paralyzed, but the knee-jerk and the plantar reflex are normal. The anesthesia will follow the distinctive distribution. As the site of the lesion descends, the muscular paralyses and the anesthetics will of course be correspondingly reduced, until its situation is such that the second sacral is spared; then we get a characteristic anesthesia which is of a saddle-shaped portion of the skin including the anus, perineum, the posterior aspect of the scrotum and penis, a small strip of skin running from the perineum along the postero-internal aspect of the thigh, but producing no anesthesia below the knee. The urethral and vesical mucous membranes are anesthetic, the bladder and rectum are paralyzed, the anal reflex is lost; there is no disturbance of the knee-jerk, or the plantar reflex. Lesions of the lower roots of the cauda equina give a more limited palsy as the lesion is lower and lower in height. First the genital apparatus will escape, next the bladder and rectum, until we come to lesions affecting only coccygeal nerves, when there will be a cutaneous palsy of the anus and perineum, but the sphincter will be intact.

DIAGNOSIS. Between the involvement of the whole cauda equina, and of the segments of the cord from which these nerves would arise. There is no difference in the distribution of the palsy, but it is lower neuron in type if the lesion is in the cauda equina; that is, there is a muscular palsy, atrophy, the reaction of degeneration, absence of knee-jerk, and normal bladder reflex, vesical and anal reflexes, with constant dribbling of urine. If the lesion causing muscular paralyses and anesthesia in a similar area is located in the spinal cord above the segments of origin of these nerves, instead of in the cauda equina, we shall have an anesthesia, and a muscular palsy, but no atrophy, no reaction of degeneration, the knee-jerk will be increased, there will be ankle clonus, and Babinski's reflex, and we shall have reflex urinary incontinence, there will be retention of urine at first, and as soon as the lower centers recover from the shock of the initial lesion the bladder will empty itself when full, without the knowledge of the patient. If the lesion is in the segments controlling these actions, it cannot

be distinguished with certainty from a lesion of the same fibres in the cauda. If the conus is affected we shall have the muscles of the lower extremities intact, anesthesia in the saddle-shaped area referred to, and loss of sexual power; also Babinski's reflex and a lack of rectal control, but the onset of the motor palsy will be very rapid, and the atrophy especially so, with the result that there is a great tendency to bedsores. There will be no pain, but partial sensory lesions.

PROGNOSIS AND TREATMENT. The prognosis of a lesion of the cauda is better than one involving the cord, since the nerve fibres which make up the cauda are capable of regeneration, while the tissues of the cord itself are not replaced after degeneration. Syphilitic exudates are more tolerable in this position, and hemorrhages are more liable to absorption. If the compression is from new growth of any kind, it is in a position where removal is among the possibilities, and whatever disease or injury may have been suffered by the bones in this locality can be detected by X-ray, and is not beyond the reach of surgery.

THERAPEUTICS. As has been indicated, the treatment for most conditions affecting the cauda equina are surgical, but in case of hemorrhage we may promote absorption by Hepar sulphur, Graphites, or Silicea, while if the case is syphilitic, specific treatment is much more efficacious.

NEURITIS OF THE PHRENIC NERVE.

This nerve arises from the 3d, 4th and 5th C., and is not liable to any form of injury, except in the case of deep stab-wounds, or injuries from bullets. While it is not a probable cause, phrenic paralysis may arise from spondylitis, and fractures, and dislocations of the cervical vertebrae. It is also possible from tumors, and aneurisms in the neck and thorax, and from hemorrhages in the spinal canal, and the different forms of meningitis, syphilitic meningitis being a quite frequent cause. The most usual cause is a disease of the spinal cord, or its envelopes. From traumatism and pressure usually results a unilateral form, but from intoxications, as from lead, rheumatism, as a factor in the multiple neuritis from alcohol, and after diphtheria, it is bilateral. Cold has been urged as a possible cause, and so has hysteria.

DIFFERENTIAL DIAGNOSIS. The diaphragm may be immovable, from Injury to the Phrenic Nerve, from Disease of the Spinal Cord, from a Myositis set up by a contiguous pleuritis or peritonitis, and from Hysteria.

SYMPTOMATOLOGY. There are evidences of paralysis of the diaphragm. When the paralysis is unilateral, its detection is very difficult, yet may be discovered after repeated examination. Even when the paralysis is bilateral, there is likely to be little embarrass-

ment of respiration, except on exertion, when respiration will become labored and the voice will become weak. On inspection it will be noticed that the thorax is in very exaggerated action, and that, on inspiration, the epigastrium will actually recede, instead of advancing. The absence of Litten's sign can often be appreciated. This consists in the fact that on inspiration a shadow will be seen to run downward from the lower border of the sixth rib, and ascend again on expiration. The observer must stand so that the bared abdomen of the patient is between the observer and a strong light. Another set of phenomena follow a loss of power in the diaphragm. There is difficulty in expectoration, defecation, sneezing, and other operations requiring abdominal effort. The sensory disturbances are obscure, and are overlooked or misconstrued as denoting neuralgia, or muscular rheumatism.

DIFFERENTIAL DIAGNOSIS. In Injuries to the Nerve, the diaphragm suffers alone, unless the neuritis is a part of a multiple neuritis. When the nerve is affected by a neuritis, there is a painful spot on the scaleni between the bellies of the sterno-cleido-mastoid, or external to them.

If it is from Disease of the Spinal Cord, other muscles always suffer, and the associated condition is often overlooked, because these muscles are those which are innervated from the cervical, and not from the dorsal cord.

If it is a Myositis from pleuritis or peritonitis, we shall find the symptoms of the primary disease, the action of the diaphragm will elicit the characteristic pain, and fever will be present.

If from Hysteria, in which condition patients will often breathe with great rapidity for a long time, even in conditions of rest, careful watching will, after a time, detect a normal movement of the diaphragm. One such movement will settle the diagnosis. We shall often be able to find the stigmata of the disease, and, in any case, ether narcosis will clear up the condition.

PROGNOSIS. It is good, in cases when evident pressure may be removed, or the results of acute injuries are amenable to treatment. It is also good in rheumatic and post-diphtheritic cases, but less so in the alcoholic. It is positively bad when it depends upon disease of the spinal cord.

TREATMENT. In a recent neuritis, we may apply counter-irritation over the nerve at the lower and inner part of the anterior triangle of the neck. Electricity is not considered to be of much avail in cases of any considerable gravity. If it is to be used, one pole (anode) should be pressed deeply into the structures outside the lower part of the clavicular portion of the sterno-cleido-mastoid muscle, while the other pole is placed upon the sternum at the border of the epigastrium, or over the diaphragm on the same side as the electrode.

THERAPEUTICS. The same as in other forms of neuritis.

MULTIPLE NEURITIS.

POLYNEURITIS, PERIPHERAL NEURITIS, SYMMETRICAL NEURITIS.

DEFINITION. This is a condition where many nerves are attacked in rapid succession by a degeneration of their axis-cylinders, and most noticeably at their terminations in the extremities. While this process extends toward the spinal origin of these nerves, it commonly ceases before reaching the nerve-roots, but exceptionally may involve the spinal nuclei, or some of the cells of the cortex. It perhaps will finally be termed a general parenchymatous degeneration possibly of the whole nervous system, but with a marked predilection for the peripheral portions.

THE SYMPTOMS OF A TYPICAL CASE. There is some degree of paralysis of all the extremities, with wasting, and later contraction, of certain groups of muscles innervated by the musculospiral and anterior tibial nerves. This paralysis will be preceded by tremor and sensory changes, or accompanied by them, and whatever be the extent or distribution it will be symmetrical. The sphincters remain normal.

DIFFERENTIAL DIAGNOSIS. To be differentiated from Neuralgia, Tabes Dorsalis, Anterior Poliomyelitis acuta, Landray's Paralysis, Myelitis, Hysteria, Spinal Hemorrhage, Spinal Meningitis.

AGE. Twenty-five to thirty-five. Children furnish but a small per cent. of the cases, being immune to all causes but diphtheria, or arsenic, and occasionally malaria or measles, while old age suffers but rarely, and then from arterio-sclerosis, atheroma, and diabetes. Thirty to forty, most common age of alcoholic form. Herter details a case in a boy three years of age arising from a drink of whiskey.

SEX. About equally when all causes are considered; but from alcohol alone, the commonest cause, the female preponderance is great. Males more rheumatic and toxic forms.

ETIOLOGY. In assuming a certain causation we must remember that the disease is very often the result of several factors. In no disease is the combined potency of a predisposing and an exciting cause more noticeable. A cachexia, or a tolerable degree of toxemia may be rendered intolerable by the added element of privation, exposure, or overexertion. To estimate, therefore, truly, the causative element to be combatted, it seems to me that these three factors just mentioned should, once and for all, be relegated to the minor rôle of exciting causes only, notwithstanding the opinion of competent observers, that at times no other cause can be discovered.

The cases fall into three classes: those due to toxic agents; those due to acute infection; and those due to a generally diseased condition of the body. In the toxic class the most potent is alcohol, its victims outnumbering all other causes combined. Not from occasional excess, but from steady use of the stronger liquors. Next is lead, then far behind is arsenic, then zinc, mercury (denied by many), then phosphorus, bi-sulphide of carbon, oxide of carbon, ergot and silver. Arsenic given therapeutically has been a classical cause, but lately many protests against its potency have been recorded. It is, however, recorded incidentally that an Italian physician produced many cases, and of great severity, in an attempt to jugulate a group of cases of chorea by doses of *Liquor Potassii Arsenit.* larger than are commonly used. While it may be said that properly regulated doses of arsenic rarely have been known to produce this unfortunate sequel, in combination with moderate amounts of alcohol it is very potent. Such are the cases of an epidemic form traced to the use of beer in which arsenic, as an impurity of glucose, has been mixed. Osler mentions a case from the use of large doses of the sulphocarbonate of zinc for two years.

OCCUPATION. Plays a large rôle in the case of painters, plumbers, type-setters, rubber-workers, match-factory employees, white-lead makers, lead-miners especially, all workers in lead, arsenic, mercury, and dealers in spirituous liquors who are apt to indulge in them to excess. More than one cause may be operative, as a consumptive may become alcoholic, or physical or mental depression in an alcoholic may precipitate a neuritis.

The acute infections are potent in this order: diphtheria, small-pox, variola, typhoid, post-operative conditions from posture and narcosis (I saw a case after laparotomy), the puerperal state, puerperal septicemia, malaria, acute rheumatism, beri-beri, influenza, the exanthemata. The effect of these may be immediate, but more often during convalescence, or a week or two after it, notably so in the case of diphtheria.

Conditions which deprave the general health of the body. Arterio-sclerosis acts by gradually reducing the peripheral circulation; syphilis, tuberculosis, leprosy and diabetes all reduce the general health, and also specifically attack the nerves at the point where nutrition is weakest. Epidemics having the character of beri-beri have been noted in Connecticut by Hammond, and in Hamburg by Eisenlohr.

PATHOLOGY. Cases have been found with no ascertainable lesion, and the present state of our knowledge does not always enable us to determine the cause from the character of the lesion. The most usual effect is a degeneration of the axis-cylinders which is most evident at the periphery, and its intensity decreases from thence toward the cord. Wallerian degeneration is set up, and

secondarily the endoneurium and perineurium are affected more or less according to the intensity of the process, with the result that, in cases of long standing, there is some grade of swelling of the nerve-trunk. In an ordinary neuritis the process is reversed, in that the interstitial tissue is first affected, and the axis-cylinder only as a result of the primary changes in the interstitial tissue. It would seem probable from the primary implication of the axis-cylinder at its peripheral extremity that the actual injury was to the cells of the anterior horn, and that a weakened cell displayed its debilitated condition by the death of its axon at the point where a diminution of nutrition would be most evident. In cases secondary to constitutional diseases, such as tuberculosis and leprosy, changes of a specific character have been found in the nerves. The cells of the anterior horns have shown changes in diphtheritic and alcoholic cases, the conduction tracts, both motor and sensory, and the cells of Clarke's column have been found to be degenerated. These changes are sometimes inflammatory, but generally degenerative, and a case of multiple sclerosis has been noted subsequent to a diphtheritic polyneuritis. The cellular changes are often inflammatory, and at times hemorrhagic. Diphtheria affects the brain, the cerebellum and the cord, and yet some cases, while widespread in their phenomena, have revealed no changes at the autopsy (Hosche). Lead, mercury almost solely, and alcohol act upon the cortical cells. Diphtheria frequently affects the ocular muscles. The optic nerve, is often affected, and alcohol is the most frequent cause of toxic amblyopia. All the oculomotor nerves may be affected. When we find that a certain disease is symmetrical and widespread, we are forced to conclude that it is due to the action of some poison which is universally distributed throughout the system, and that is equivalent to saying that it is in the blood.

GENERAL SYMPTOMATOLOGY. All cases are common in displaying to some extent Disturbances of Sensation, Paralysis, Incoordination.

Sensory Changes are first in time of appearance, and also the last to go away so far as ordinary investigation or the patient's observation informs us, but Papoff asserts that faradic reaction is the earliest symptom, and Church says that a diminished knee-jerk has preceded by weeks the apparent onset of the disease. The history of a case will generally reveal that the patient began to notice sensations of numbness, creeping, crawling, stinging, burning, chilliness or heat in the extremities (paresthesias). They will be confined to the extremities for a few weeks, and then extend to the forearms and legs. They are not in stripes, or irregular areas, as in cases of simple isolated neuritis, but cover the extremities like a glove or stocking, reminding one of hysteria. They are not found upon the trunk, and literature furnishes only one

case of an invasion of the face. This is the stage of nerve irritation.

Next we find that the nerve-trunks have become swollen and tender to touch, the muscles and joints as well, and there are spontaneous pains and cramps. These latter are most pronounced in the lower limbs. This is the stage of activity in the process of nerve-degeneration which secondarily sets up an interstitial inflammation in the connective tissue of the nerve-trunks, and also some ill-defined changes in the muscular elements.

Next is a loss of all tactile perception, and often for all varieties of cutaneous sensation, heat and cold, and pain, as well as ordinary touch. This is the stage of completed degeneration. An element in this loss, at times, is a diminution or abolition of the joint and muscle sense, originating according to Batten in a degeneration of the muscle-spindles, from which run fibres in the sensory portion of spinal nerves to arborize about the cells of Clarke's column in the cord, whence new axons go up in the direct cerebellar tract to the cerebellum. This mechanism presides over static equilibrium, and hence we may find total ataxia with, or without much, paralysis. This is the pseudo- or neuro-tabes of the French, and often confuses this disease with tabes dorsalis, whose ataxic symptom is based upon a chronic form of this same degeneration.

Throughout all these changes, and final loss of sense perception, there runs an undercurrent of abnormal sensibility to pain, and therefore while the patient may fail to recognize the touch of a finger, or wince at the prick of a pin, he may complain bitterly of the touch of a feather, or of lumps in the softest bed, or of knots in his clothing. It is *anesthesia dolorosa*, or painful anesthesia. This is most noticeable in poisoning by arsenic, and least in cases from lead, while alcohol presents most sensitiveness along the trunks of the nerves. Alcohol and grippe give rise to the most spontaneous pain. Pains about joints and in muscles are found in other diseases, and care may be needed to assign to them their true origin, but with care it will be found to be most pronounced along nerves which are distinctly swollen, and this with a symmetrical distribution is quite diagnostic. The area of these sensory changes conforms quite closely to that of the motor losses, and it varies quite considerably during the course of the disease. Convalescence is signaled by tingling and other sensations in parts that had become insensitive, and is usually perceived in the part last affected, and pains and over-sensitiveness may persist for a long time.

Motor Symptoms. The motor loss is usually rather relative than absolute, and would be of the grade termed a paresis rather than a paralysis. Rarely it may be sudden and very profound. The nerves typically affected in the upper extremities are the

terminal branches of the musculo-spiral, which are the radial and the posterior interosseous supplying the following muscles: Those of the posterior aspect of the forearm; extensor carpi radialis brevis, supinator (never in lead palsy), extensors (all the surrounding ones), branches to the carpal joints. These muscles are the extensors of the wrist and hand.

In the lower extremities the lesion is of the anterior tibial branch of the sciatic which supplies the following muscles: tibialis anticus, extensor longus digitorum, extensor proprius hallucis, peroneus tertius, and through the internal and external branches; the external to the extensor brevis digitorum and the tarsal joints. Internal unites with musculo-cutaneous nerves. The office of these muscles as a whole is to dorsally flex the foot, and to extend the toes. From these losses we get the characteristic wrist and footdrop. These nerves are nearly, but not absolutely invariably the ones attacked, as in the arm we sometimes see, although in a very small per cent. of the cases, the deltoid, innervated by the circumflex, the biceps and long supinator, and the spinati affected first. The median nerve may be affected as well as the circumflex, and in such cases we find a distinctive deformity in "Claw-Hand." In the lower extremity the calf muscles, internal popliteal, and posterior tibial nerves may be paralyzed, and then the heel will strike first in walking, the foot will be inverted, and there will be dorsal flexion. Sometimes erratically the muscles of the eye may be affected, or the diaphragm may be paralyzed. In a marked case the foot drop is complete, and while the patient is lying down the foot tends to the position of equinovarus. Milder cases are unable to dorsally flex the foot beyond a right angle. The toes are flexed and bunched, and can neither be extended nor separated. Muscular weakness can be estimated by their reaction to resistance. The gait is characteristic, as the thigh must be raised to flex the foot, the foot is then thrown forward by swinging the leg like a flail, the outer border of the foot is depressed, and the pendent toe strikes the ground. The outer side of the foot may be the first part to strike. The other foot is then swung around in the same way giving a high knee-action, the "steppage gait" of Charcot. To continue the variations of gait, it may be stated that if the thigh muscles are paralyzed or greatly weakened, a possibility in alcoholic cases, walking may be impossible, or well-nigh impossible.

While distinctively a peripheral disease, it is not always so, but it is a possibility that every voluntary muscle of the body may be implicated, and the viscera even may be attacked. The trunk muscles are affected after the peripheral ones have been severely damaged. In such cases standing, walking, or even sitting may be impossible. In very widespread cases we may find implication of the muscles of the eyes, face and neck, of the tongue,

throat, larynx and diaphragm, of respiration and of the heart. The patient may have to keep awake for long periods of time to make respiration possible. Such cases most usually flow from the effects of the toxins of diphtheria. The symmetrical character of the disease is especially well-marked in the muscular palsies, although it does not imply that the extent of such palsy will be the same in both limbs, for it is practically an axiom that the initial member is more deeply affected than its fellow which is attacked at a later date. The progress of the paralysis does not proceed according to any rule except this, and it is a very essential one to bear in mind, viz., the trunk is not affected until after the limbs. As an ordinary rule it is one forearm and then the other, and then one foot and then the other, or beginning in the feet and after affecting both, then, and then only, extending to the hands, but the rule is not by any means an invariable one. There may be a paralysis of a few muscles in one hand, and then a foot may be invaded, but however wide or narrowly circumscribed is the process one is struck with the implication of a whole set of movements, which points to the nuclei in the cord or bulb as the actual seat of the damage, since combined muscular action, constituting movements, and not muscles, is represented in the spinal segments and cortex of the brain. Lead and diphtheria are most efficient in causing motor paralysis.

Reflexes. As a general statement it may be said, that from the outset the deep reflexes are greatly diminished (a primary exaggeration is possible, but rare), and the superficial are almost certainly so. Speaking exactly, it must be said that it all depends upon the degree of injury of the nerves, and the location of this injury. Just so long as any intact fibres remain in the nerve, there may be some reflex; and hence it may disappear in the knee, and be active in the wrist and elbow, but it is so nearly a universal truth that the extensors of both the upper and lower limbs are degenerated at the same time, that we may confidently look for a general reduction of all the deep reflexes to extinction, or nearly to that degree. The superficial are more universally absent, but Purves Stewart notes that in a case characterized by extreme hyperesthesia of the soles there was an exaggeration of the plantar reflex, and an exaggeration of tendon reflexes. The organic reflexes, those of the bladder and rectum, are not impaired. Sometimes they will appear to be so in the last stages of very severe cases, but examination will show that it is only in cases where the mentality is so impaired that the calls of nature are not comprehended.

OPTIC NEURITIS. Occasionally it is associated with facial palsy. It is unusual in multiple neuritis, but it is occasionally found, and is of the retro-bulbar type preceded by decided choked disc and papillitis.

Muscular Atrophy. This is a variable symptom, and yet the degeneration of the tissue of a muscle follows and keeps pace with its loss of power. It is localized in the same muscles, if present, as the paralysis. Fibrillation is present. There are cases where there is little atrophy, while there is great loss of power, and the reaction of degeneration is present. Voluntary muscles, and even viscera may be affected. Owing to muscular losses, a disuse atrophy may affect other portions of the body. Trophic conditions may be altered in any of their forms, as in vaso-motor control, irregularity of the secretions, both internal and external. The hair, and nails, and skin are apt to show changes, and there is general malnutrition. Sometimes it will be limited to glossy skin of the extremities, and an emaciation of the tips of the fingers, such as we see in wasting diseases.

Contractures. These are very apt to occur, and if present will prove to be a great source of disablement, even if the power returns to the limb, and should therefore be antagonized at the earliest evidence of approach. They arise from four causes:

1. Lack of balance of the muscles on the two aspects of a limb.
2. In part from a natural predominance of the flexors.
3. In part from the action of gravitation.
4. In part from muscular fibrosis, and fibro-tendinous contraction.—(CHURCH.)

From this condition arises ankylosis of the hands and feet, elbows and wrists, and even the hips and shoulders, and tenotomies may be needed to allow the heel to touch the floor.

Tremor. From the lack of muscular balance, and a general loss of muscular strength, we are apt to get all grades of tremor, to which symptom the loss of joint and muscle sense is also contributory, and they also produce the ataxia and incoördination which have already been referred to. It may show itself only as a clumsiness in movement. Almost always present, and may antedate the motor palsy (lead particularly), elicited only by attempts at motion, and most notable in arms, lips and tongue. This is marked in cases caused by alcohol. Especially evident in lips and tongue, and muscles of the face also. In lead it comes early, and may last as long as the paralysis; in cases from mercury, rarely from arsenic or opium, and may also come from tobacco.

Electrical Changes. At different stages of the same case we may find exaggeration in the outset to all qualities, and after a short time the reaction of degeneration, or there may be simply a reaction to galvanism, with a loss of response to faradism, which even in the severest cases is very rarely total. Erb mentions a case in which there was increased galvanic response; a myoidema lasting for several minutes. Osler quotes from Starr. Frequently faradic reaction alone is lost, but it may be a simple reduction of reaction to all qualities. It is a pathognomonic, that when the

muscle alone reacts to galvanism it takes a strong current to produce any contraction, while in anterior poliomyelitis a weak current will cause a response for several months. Loss of faradic, and lowered response to galvanic, is a sign of multiple neuritis. Anodal closure contracture equal to, or more potent than cathodal closure contracture is the reaction of degeneration.

Mental Disturbances. A moderate amount of mental disturbance is very common in neuritis, and when slight it may be overlooked. It is most frequent among alcoholics; next in frequency come those caused by lead, and the infectious cases least of all. It may precede the onset, but generally develops along with the disability. This psychosis was first commented on by Starr, but in the year following, Korsakoff described it very fully, and alleged that it was peculiar to alcoholic cases. This has not been fully sustained, and the opinion held at present is, that upon a neuropathic heredity a confusional state might be developed by toxic substances, or by some acute infections. It is characterized initially by a change in the disposition of the individual, who shortly becomes a victim of insomnia. He then becomes subject to vague alarms, which at first possess him only by night. He may then become maniacal, and have hallucinations of sights and sounds, and of the special senses, after which he becomes depressed. The maniacal stage may be lacking. In any case there is a weakness of memory, and forgetfulness concerning, however, the near past alone. Under such conditions, misled by his hallucinations, he becomes utterly untrustworthy in his statements, and he will relate fictitious events with a wealth of detail. He shows a great lack of the power of concentration, and Oppenheim states, of orientation also. This is the power of correctly estimating his position in relation to the world surrounding him. Starr states that the depressive condition is peculiarly distressing, because his memory is too feeble to permit him to retain the consolatory admonitions of his friends and attendants, but it is to be diagnosed from typical melancholy by the fact that he does not indulge in self-recrimination as the author of his own misfortunes. Death may ensue from the gravity of the insomnia, or the violence of the cortical disease.

ALCOHOLIC NEURITIS. This is the most usual form, and is most constant in its symptomatology, and comprises more of the characteristics of multiple neuritis than that from any other cause. Lower extremities are most affected. Invasion is usually sub-acute. Pain is more pronounced than in any other toxic form. Usually after an attack of gastritis, or exposure to damp and cold, the patient may develop a weakness in the lower limbs, which increases so rapidly that at the end of a week he may not be able to walk at all. Paralysis is less marked than in the others. Paresthesia is most intense and intolerable. The muscles are

very sensitive. Cutaneous hyperesthesia is exquisitely developed in the palms, soles and tips of the fingers. The quadriceps extensor is attacked, as well as the extensors below the knees, and hence the high-action or "steppage gait" of Charcot is well marked. Atrophy is prominent, and fibro-tendinous contracture is pronounced. Ocular palsy is common. Vision is principally if not alone affected (amblyopia); it is bilateral, symmetrical, and insidious in approach, and must be searched for, as the patient may be ignorant of it. He may unconsciously have pronounced papillitis, retracted visual fields, color scotomata, and considerably reduced vision, and be ignorant of it. Squint is not common, but may be present; it may be due to an oculo-motor neuritis and this is usual, and then the muscles are affected singly. It may, however, affect all at once. In this case it is due to nuclear involvement, and is usually accompanied by apoplectic features. Rarely dilatation, contraction and inequality of the pupils have been seen, but the pupillary reflex is not disturbed, and the Argyll-Robertson pupil has not been observed. Mental disturbance is most common in multiple neuritis from this cause. Recovery is rapid under total abstinence from intoxicating liquors. Relapse is rapid and striking from the least indulgence. It may be without fever, or quite slight, or it may be pronounced. If a secondary rise of fever occurs it is often the sign of a pneumonia which is quite likely to be overlooked, as it is apt to be irregular in its localization. At this time the patient will be found to be tender to touch, and this tenderness will seem to be seated in the muscles rather than in the nerves, but if the trunk of the nerve be followed up, it will be found that it is the site of the greatest tenderness. The knee-jerks will be found to be diminished, but they will never be completely lost, under careful examination, since some fibres of the nerve are always preserved. The muscles of the trunk generally escape, and if attacked it will be a very late symptom, (the reverse of what occurs in Landray's Paralysis). The affected muscles waste, and contractures occur, but they differ from the same symptoms occurring after a hemiplegia in that they are generally curable by care, and patient treatment. The muscles show the reaction of degeneration, and also a phenomenon which is diagnostic, viz., if a muscle be tested with an electric current, the resulting pain lasts for hours, while under ordinary conditions we should find it to be only a momentary distress. While the deep reflexes are variable, the skin reflexes are quickly diminished. Tremor is almost always present, and is widely distributed, but almost always spares the face. It is often only visible on attempts at muscular action, and is most evident in the arms, lips and tongue. Jactitation of the lips is insisted on by Osler. Mental disturbances have been thoroughly discussed in the general symptomatology.

DIPHThERITIC. It is clinically more common in adults, probably from the fact that its common manifestations as a pharyngeal, laryngeal, and pneumogastric paralysis are so rapidly fatal in an infant that the death is hardly ascribed to the nerve-lesion. The throat and palatal muscles are most affected here, and at times exclusively, and as a result the patient may be embarrassed in swallowing, and the voice has a nasal quality, and food regurgitates through the nose. There are some cases which display a sensory and motor type of palsy like other forms of multiple neuritis, and this form is apt to be fatal, as it has an additional predilection for the nuclei of the medulla. The preceding illness is so recent, that it often guides us to the correct diagnosis, as the neuritis generally appears in from one to three weeks after the subsidence of the original malady, but may be present within a day or two of the appearance of the pseudo-membrane, or may occur in absence of this condition, but where the original disease has been so slight as to escape notice, the primary implication of the muscles of deglutition is a diagnostic point. The muscles of the soft palate and pharynx are the first to suffer, lips and cheeks may follow, the tongue also, and the legs next if it extends. Knee-jerks are often abolished before even the palate is invaded. The muscles of the neck are often so weakened that the head rolls about. The upper extremities are rarely invaded, wasting of the muscles is rare, contractures seldom develop, sensory damage consists of insensibility in the parts; severe pain and painful paresthesias are wanting. Ataxia to some degree is usual, Romberg's sign is seen, paralysis of accommodation occurs, so that there is loss of acuteness of vision for near objects, but there is no distinct pupillary paralysis. It comes at the time of the palatal paralysis. In children we may test by the use of a pinhole in a card, and in adults by print. In both cases the outline will be blurred. Church calls it paralysis, as he says that the neuritis cannot always be demonstrated. Hochhaus found only the muscles affected. Babinski paralyzed rabbits with the toxin from the Klebs-Loeffler bacillus, and found no changes whatever. In certain cases a peri-axial neuritis is plain. Why in some, and not in others, is not clear, but in all cases it is certain that the symptoms are due to toxins developed in the life history of the bacillus. The palsy may appear after any sort of a case, without regard to the location of the initial malady. It may follow from simple exposure which has given no evidence of its presence by sore throat or pseudo-membrane. Strabismus, double vision, and ptosis are not uncommon. Sexual impotence is frequently developed in males of adult years. Mental symptoms almost never occur. Diphtheritic paralysis is rapid in development. Recovery is usual. If the palate alone is affected, recovery is usual in two to three weeks; if generalized it may last three months to a year.

Rarely, paralysis is fleeting, and may be recurrent. Death is from inanition, cachexia, pneumogastric paralysis, inspirational pneumonia.

MERCURY. This is more an affection of the brain displayed upon the nerves than a primary affection of the nerves themselves. The motor and sensory palsies are present, but they are not so marked as the tremor, which is the prominent symptom. This tremor begins in the face, then goes to the trunk, and then into the arms and legs, and while at first it is only apparent on attempts at movement, it finally becomes constant. Lead may also show a like tremor, but it is in connection with colic, and the development of a blue line on the gums, while with mercury we find a stomatitis, and a swelling and soreness of the gums, and a loosening of the teeth from a gingivitis.

ARSENIC. This is a very general form of paralysis with a mixture of motor and sensory symptoms. The finer muscles of the hand are more often affected than in the other forms, and it is most evident in the legs. Ataxia is prominent, and there is considerable atrophy. The cause can generally be found in arsenic taken as a poison; as a medicine (very rarely), or, at times, from gradual absorption from fabrics, or wall-coverings.

Osler doubts the effects of drugs as ordinarily administered, and is not sure that it can arise from the emanations from fabrics and wall-coverings, since the Styrian peasants have been known to take 8 grains of arsenious acid a day. It is as poisonous in a volatile as in a solid form, and if derived from fabrics it is from its transformation into a volatile form by the action of certain mould fungi. It has also been proven that dampness, and a temperature from 60 to 95 degrees Fahrenheit were sufficient to volatilize it. He had a case which developed a multiple neuritis from the effect of arsenic taken for the cure of Hodgkin's Disease. The patient took 4 ounces and 1 drachm of Fowler's Solution in 75 days, and during that time omitted 14 days, so that in fact he took 32.4 drops a day while actually taking the drug.

The resulting palsy is like that from lead, except that the legs are particularly affected, and in them the extensors and the peronei, so that the patient has the "steppage gait." Early in the case, if one asks the patient to extend the wrist, and spread out the fingers, a tremor will be manifest. Reaction of degeneration may precede the palsy. From arsenic, symptoms like those of erythromelalgia have been seen. There is ridging of the nails. Marked tremor is rarer in arsenical poisoning than in any other, except that from opium. Dark mottling of the skin is diagnostic. In testing for the drug remember that the urine of healthy persons will show the reaction for arsenic in 30% of the cases.

LEAD. This intoxication usually arises from prolonged exposure to the metal through the use of liquids contaminated with

lead from pipes, or containers, or cosmetics, or drugs, and is quite apt to be unsuspected, but may be accidentally developed into a neuritis by the administration of alkaloids. Sources of lead may be hair dyes, face powders, styptics, injections which contain lead (all may delay in their action), fabrics, wall-papers, toys, medicinal preparations. A person may as ignorantly take lead into the system, as a person may alcohol in using a remedy said not to contain alcohol. It ought to be suspected in one who is engaged in an occupation in which lead is extensively handled, and even in the absence of such a history, if the neuritis has been preceded by obstinate constipation and abdominal colic. In chronic poisoning the muscles will show fatty degeneration, and fat in yellow patches. While the nerves show peripheral degeneration, the cord and nerve roots do not, and Osler does not say that any degeneration of the cells of the anterior horns has ever been found, but the hypothesis is that where there is primary degeneration it is due to such change.

Anemia marks all of these cases, and Cadwallader says that nucleation of these cells, i.e., the red blood corpuscles, is present in even the slightest cases, and is diagnostic. The corpuscles are not reduced however, in even the severe anemias, below 50%. Grawitz first, and then Pepper, White, and Vaughan, have found that it is diagnostic of lead intoxication to find with this moderate reduction of the red corpuscles a granular deposit in the cells, which is baso-philic to Jenner's stain and also to polychrome methylene blue.

The diagnostic blue-line is of two kinds. The first is between the gums and the teeth, and can easily be scrubbed off, while the other is a sulphide of lead deposited in the capillary loops, and with a magnifying glass can be seen to be in transverse lines, and not in a solid mass. It may be permanent or short-lived.

It is the most exclusively motor of all the forms of neuritis, and while there may be some pains in the arms, and some cramps, there are usually no other disturbances of sensation, unless the legs be affected, and then changes are below the knees. Pains in joints and legs are common at the outset, but there may be no sensory features at all. The upper extremities are almost exclusively affected, and certainly are the first parts to be attacked, and the hand most used will be the point of onset, but the paralysis will later become symmetrical. The site of invasion is the common extensor of the third and fourth fingers (the common extensor of the fingers is innervated by the posterior interosseous branch of the musculo-spiral), then of the wrist and then of the little finger. From this distribution we find wrist-drop to be the characteristic deformity, and if it extends upward, the supinator is commonly exempt, since the nerve especially affected is the radial branch of the musculo-spiral only. In severe cases we may

find it extending to the muscles of the arm and the shoulder, later perhaps the legs, and in some cases the muscles of the trunk. Even when the trunk muscles have been affected, the prognosis is better than in the alcoholic type.

Fibrillary twitching ensues, and then prominent wasting. This may precede the palsy like a myopathy. Tremor is one of the commonest signs of lead intoxication. Retrocarpal tumor (Gubler's tumor) is frequent (no importance).

Madame Klumpke-Dejerine has divided the peripheral effects of lead into the following groups:

Ante-brachial type. Extensors of fingers and wrist involved, and therefore it is from the musculo-spiral. The supinator usually escapes. A swelling occasionally occurs on the wrist, which is known as Gubler's tumor, but it is of no importance.

Brachial type. Deltoid, biceps, brachialis anticus, supinator longus, pectorales rarely. This form is bilateral.

Aran-Duchenne type. Small muscles of the hand waste, also thenar and hypothenar eminences. Atrophy is marked. It is common in tailors, and may be the first manifestations of lead palsy.

Peroneal type. There are 100 cases of the arm type to 13 of the leg type. The lateral peroneal is affected, giving rise to palsy of the communis digitorum, and the extensor proprius hallucis, and gives rise to the "steppage gait."

Laryngeal form. This has been noted by Morell Mackenzie and others. It is a palsy of the adductors of the larynx.

Atrophy of the Scapulo-humeral type. This may follow the first form, or be primary, and is bilateral. It may be generalized, and begin in the wrists, and may involve every muscle below the neck.

Aphonia is common, as the laryngeal muscles are often attacked.

It sometimes sets up an optic neuritis, and sudden blindness may occur from this.

Mental disturbances may be like the general type of psychosis common to all the varieties of polyneuritis, or one peculiar to lead intoxication. This is an acute mania, which with the bodily symptoms reminds one of general paresis, but differs in the fact that expansive delusions are replaced by mental sluggishness. The common conditions caused by lead are delirium, convulsions, coma, hemiplegia, and all these in varying combination. The poison may induce arterio-sclerosis, and from this may arise lesions of the kidney, and this in turn may set up cerebral disturbances. This may be the cause of the mania.

DIABETIC. Neuralgias are apt to occur in the course of this disease, in consequence of the toxic condition of the blood, and also of the anemic condition. This engenders an enfeebled nutrition of the nerve, and the sciatic is the one most often involved.

Multiple neuritis from this cause is exceedingly rare (Starr), but it is exceedingly interesting because it so closely resembles *tabes dorsalis*. Thirty per cent. of all diabetics lose their knee-jerk (von Ziemmsen, quoted by Starr), and they show ataxia and pains of all kinds, and also perforating ulcer. These phenomena seem to bear no relation to the amount, but to be dependent upon the mere presence of sugar, although with the reduction of the sugar the symptoms of neuritis pass away. Lack of Argyll-Robertson pupil differentiates it from *tabes*.

MALARIA. This has been known to cause multiple neuritis, but the outbreak has always been subsequent to an attack of malarial fever, or has shown its character by being promptly suppressed by quinine. It is peculiar in leaving the hands unaffected, and by attacking in the legs (the anterior tibials), the flexors of the ankles and the extensors of the toes. There is hyperesthesia.

RECURRENT MULTIPLE NEURITIS. Sherwood, Ross, Dreschfield, Targlowa, Klumpke and Osler have described such a type, and Thomas has described a case. It appears that certain persons are susceptible to repeated attacks from various poisons, especially lead and alcohol, and this without renewed infection from the poison. Whether this is due to an original super-susceptibility, or to the prolonged effects of the original toxemia is not known.

BERI-BERI, OR KAKKE. A polyneuritis from a specific infection. No organism out of the many found has been agreed upon. Some say carbonic acid gas poisoning, some to a diet of fish and rice. It certainly arises only when people are closely aggregated as in institutions, and in tropical countries where these conditions are habitual.

SYMPTOMATOLOGY. Paralytic and atrophic. The lower limbs particularly, and those of the antero-external leg muscles, giving a characteristic gait. The phrenic and pneumogastric nerves suffer early giving rise to respiratory and cardiac symptoms. The face and tongue are frequently affected; sensory disorders are mainly anesthesia and severe lightning pains. Edema of lower extremities is often great, and may flood the body cavity and invade its structures. Knee-jerk is lost early; right cardiac dilatation is frequent, with rapid heart action, and murmurs over the base. Mental disturbance like that of alcohol is frequent. (Absolutely rare, some say, and when present is hallucinatory—confusional in form, and of short duration.) All grades of severity from a simple weakness of the lower extremities with cardiac palpitation, to a pernicious acute form like an acute ascending myelitis, causing death in a few days. Intermediate varieties are always long in recovery, with a constant liability to death from pneumogastric accidents. A large proportion of cases is said to show the presence of intestinal parasites. Diagnosis is from clinical peculiarity,

and from the fact that it occurs in persons who have been living on foods low in proteid value.

LEPROUS NEURITIS. The leprous bacillus can set up a neuritis. This causes a fibroid proliferation in the nerve-trunks, which often become nodular, and at length the bacilli disappear. The spinal cord is also invaded, and presents central cavities, especially in the posterior horns, and gray commissure. Prominent sensory symptom is anesthesia in discrete plaques, favoring in location hands, feet, forearms, legs and face. By spreading, large portions of the body may become anesthetic, and the deeper parts also, muscles, bones, etc. Muscular atrophies are comparatively slight, affecting mostly the small muscles of the hands, feet, and face. Atrophic conditions of the extremities may lead to mutilation of them. Evolution may be very slow, lasting years. Dissociation of cutaneous sensation may be encountered, like a syringomyelia of the Morvan type, probably due to the invasion of the cord previously noted. This variety is diagnosed from its clinical features, and from the discovery of the characteristic micro-organism.

AUTO-TOXIC CASES. The great class of cases arising from intestinal fermentation and the infections must be diagnosed from their history. A well-marked case presents little difficulty in its recognition, since we find a symmetrical loss of power in the muscles situated first, and generally exclusively, in the extremities.

COURSE AND PROGNOSIS. In predicting the course of events it is pretty safe to assume that the disease will steadily grow worse for about six weeks, and by that time it will attain its maximum intensity, and wideness of distribution, then it will remain practically unaffected by treatment for from three to six months, and that recovery will take place in from six months to two years from the inception of the attack. Some cases are very acute, and rapidly invade the diaphragm and bulbar nerves, and are fatal in from six to twenty days. The probable duration of the attack may be predicted in a general way from the degree of its intensity, the wideness of its distribution, and the possibility of removing the cause. Recovery is the rule, even if cases have lasted a year. Diphtheritic cases are the most dangerous in the early stages, since respiration and cardiac action are more likely to be compromised, but in comparison with all other causes it is most sure of recovery. Lead, of itself, rarely is a cause of death, but, since the patient's general condition has been previously deteriorated by the poison, the disease tends to be slow and disabling. Alcohol tends to be more fatal, since the patients are apt to have been previously debilitated by the poison. It is characterized by a great deal of pain, and that tends to exhaustion; they are peculiarly liable to pneumonia, and it is often overlooked, since it is apt to have an unusual localization. Cases arising from auto-intoxication are the results of a very long-standing bowel disease,

and so tend to be of long duration, since the exciting cause is still operative. If the invasion is acute and the localization wide, the patient may die in the early stages, but, if tided over this time, is apt to make a speedy recovery; but if the onset is slow it presages a chronic course. If the sphincters become impaired, it denotes an involvement of the cord, and whatever dangers that may imply. In any case, severity betokens initial danger and prolonged convalescence, while mildness presages rapid recovery. Good general condition insures prompt recovery. Contractures are generally curable. Tenderness in the muscles may remain long after muscular power has been regained, and so delay recovery for a long time.

DIAGNOSIS. The symptoms of all kinds are symmetrical in distribution. The deep reflexes are depressed while the sphincters escape. The superficial reflexes are abolished. Sensation is always perverted; at first paresthesia and later areas of anesthesia, while often there is some degree of spontaneous pain. The nerve-trunks are tender, there are tremor and atrophy; contractures are common. There is the reaction of degeneration, and mental disturbances are not uncommon. There is the presence, or history, or some competent cause.

ETIOLOGICAL DIAGNOSIS. Gowers says that in chronic metallic poisonings the arms suffer before the legs. Where the arms and legs are affected at the same time, it is extremely improbable that the neuritis is due to a metallic poison. Stewart, in Aliehin, says that when from lead, the left hand is affected before the right only in left-handed persons. We get Gubler's tumor. An initial invasion of the proximal portions of the arms and legs, at the same time as the distal portions, denotes that the cause is not alcohol; probably cold, or a toxemia. From arsenic the affection of the arms is soon followed by that of the legs, extreme sensitiveness, and bronzing of the skin, with cutaneous affections and ulceration of the gums, while from alcohol the legs suffer first. Cold or toxemia should be suspected where the muscles of the trunk or the bulbar nerves are affected early, and if there are grave constitutional symptoms. Diabetes should be suspected when there are irregular neuralgic pains, beside those located as we should expect in a multiple neuritis. In diphtheritic cases the knee-jerks are commonly lost, even if there is no paralysis of the legs. The associated symptoms should be carefully observed for diagnostic purposes relating to etiology. No form or aggregation of symptom is, however, sufficient to certainly guide us to the cause of the attack.

DIFFERENTIAL DIAGNOSIS. No disease is more often confused with multiple neuritis than Tabes. This is not to be marveled at, when we remember that both diseases have symptoms in common, and many *apparent* similarities. Knee-jerk is lost early in

tabes; there is always a stiff pupil, and usually (90% Starr) that form of ciliary paralysis which is indicated by the loss of the reaction to light, while that of accommodation is preserved, i.e., the Argyll-Robertson pupil. It has an ataxia from incoördination alone (loss of muscle sense), since there is no actual paralysis, and the patient is clumsy and weak in his movements, because he has lost that sense by which he has been able to judge of the force and order of muscular efforts. This makes his gait a slapping one, in which his heels first strike the floor. Early he has lightning pains in the legs, and a cuirass sensation like a corset about the chest, girdle sensations about the abdomen, and less often about the limbs, while he is liable to be tortured by painful colicky spasms, momentary, or of hours in duration, in the stomach, bowels, testicles, and at times in the larynx and other parts. His nerve-trunks are, however, never tender, and atrophy is late and slight, although the joints may be extensively disorganized in the last stages of the disease, when the diagnosis has long been certain. Perforating ulcer of the foot is common. The muscle sense is lost very early; some say it is the earliest symptom; and there is incontinence of the sphincters, particularly of the bladder. These are the organic reflexes. Optic atrophy is often present, and usually some of the oculo-motor muscles are paralyzed. Some form of dementia is apt to occur, but only in the later stages. The developement is very slow, with numerous remissions which may be months or even years in duration, but the disease is progressive and possibly incurable. It is certainly syphilitic in 90%.

In multiple neuritis, on the other hand, the knee-jerk is also generally absent, but it can sometimes be found, perhaps after repeated examinations, by some method of re-enforcement. The Argyll-Robertson pupil is never found. The ataxia of typical multiple neuritis is apparent only, and is due to the advancing paralysis. Since this paralysis is selective, the resulting change of gait will differ from that of tabes in that the patient raises his leg high enough to raise the dragging toe from the ground, which would be the first part to strike, followed by the outer side of the foot. The spontaneous pains of tabes are replaced by tenderness of the nerves and muscles, and the tissues about the joints. Atrophy of the affected muscles is early and pronounced, but Oppenheim states that he has seen only two cases of perforating ulcer. While optic atrophy does occur, it is unusual, since the typical optic loss is a diminution of the normal acuity of vision (amblyopia). The sphincters, or organic reflexes, are never impaired, except as an early and transient symptom, although the patient may occasionally be so dull in mentality as to ignore the calls of nature. The mental disturbances are not very common, and if present are symptoms of the early, rather than of the terminal

stage, and are hallucinations and delusions with mania, rather than dementia. There are changes in circulation, so that parts of the extremities are livid and edematous, but the joints are never disorganized. There are no crises of stomach, bowels, testicles and larynx, but only such disturbances of the digestive organs as would naturally be expected from the usual intoxicants. The disease is relatively rapid in onset, without intermissions, unless the exciting cause has been renewed, and sets up an exacerbation, and there is great probability of cure in from two months to two years at the longest.

So long as multiple neuritis adheres to typical lines we can see, on examination, broad points of difference, but there is a class of cases called by Dejerine "neuro-tabes," and by others "pseudo-tabes," where the immediate diagnosis is very much more difficult, and at times impossible. These are the cases where there is a great amount of ataxia at the outset, with few or slight symptoms of either tabes, or multiple neuritis, and it may be either one of the two. Oppenheim points out that the confusion arises from occasional irregularity in the point of attack of tabes. While usually the peripheral nerves are not attacked for a long time after the degeneration of the posterior columns of the cord, in certain cases these peripheral nerves may suffer first. Batten has gone a step farther, and has discovered that the presence of ataxia proves that the sensory nerve-endings in the muscle-spindles are diseased. He has found it in those ataxic from tabes, and also from multiple neuritis. For clearness it may be stated thus: late in tabes we are certain to have degeneration of the sensory nerve-endings in the muscle-spindles, and it is a chronic and progressive degeneration, while it may rarely occur as an early symptom; very rarely these muscle-spindles may be affected in multiple neuritis, but only transiently; both the chronic and transient condition produce ataxia. Such cases of ataxic multiple neuritis (neuro-tabes) have been known to result from alcohol, arsenic, diphtheria, diabetes, measles, and other acute diseases. Neuro-tabes produces a rapid loss of knee-jerk, muscle-sense, and therefore Romberg's sign is present. There is optic neuritis, but the paralysis does not affect the arms. Pain is common to both tabes and neuro-tabes, and its intensity is not diagnostic. Where the pain and ataxia are both moderate in degree, the early recovery is the only ground for a certain decision. If, however, with a slight loss of muscular strength, the patient is so ataxic as to be unable to stand alone, the case is tabes. The ataxia of neuro-tabes is a mixture of sensory and motor paralysis. True lightning pains are always tabetic, while on the other hand extreme muscular soreness is never tabetic. An excessive skin reflex may be found in tabes, but it is painless, while it is very rare in neuro-tabes, and if found is painful. Pupillary rigidity and bladder disturbances are

the mark of tabes, but are never in neuro-tabes. An extreme rapidity of onset is often a characteristic of neuro-tabes, but never seen in true tabes. Neuro-tabes arises from some assignable, discoverable, recent intoxication. (Always from alcohol, arsenic, or diphtheria.) (Rarely affects the arms. Oppenheim.) Gowers says pains and ataxia are almost the sole symptoms. Diagnosis between tabes and pseudo-tabes depends upon the following: onset more acute; some infection of toxic cause can be discovered; absence of bladder disturbance; absence of pupillary rigidity; (seen only once in an alcoholic by Oppenheim, once by Eperon).

Acute Anterior Poliomyelitis usually attacks children who have previously been well; the onset is abrupt, and, while it may be ushered in with fever, it is neither prolonged nor recurrent. The resulting paralysis is immediately apparent; it is at first a general paraplegia, or hemiplegia, or monoplegia. This condition of generalized paralysis soon passes away, leaving a relatively small, well-defined area of paralysis which tends to be permanent. This usually affects some group of muscles which is concerned in a definite set of movements, and is rarely symmetrical on the two sides of the body. The sensory symptoms are slight, affecting the back, or the muscles which are to be permanently affected, and if present at all it is at the outset only. Ataxia is never present. Fibrillation is present, and is distinctive of approaching atrophy. The cranial nerves, except by a coincident neuritis, are never affected, and there are no mental symptoms. The disease always leaves some deformity, and the atrophy follows the paralysis, although the interval may be a short one. On testing with electricity, it will be found that the galvanic response will persist for some months although it finally disappears.

In comparing multiple neuritis with this disease, we shall note that it is a disease of adults, except in those unusual cases where children succumb to the intoxication of arsenic or diphtheria, or much more rarely to malaria. There has been an evident or discoverable cachexia or intoxication for some time previous. The onset is subacute, or insidious. Fever is often prolonged, and sometimes recurrent. Muscles in different areas are affected, and the disease is extremely symmetrical. Sensory changes are most marked in the nerves and muscles, and they persist in some form as long as the disease lasts, and sometimes longer, and there are cramps in the muscles, which latter never occur in acute poliomyelitis. Ataxia is common, but there is no fibrillation of the muscles, the cranial nerves are frequently affected, and mental changes are rather common. Deformity of a permanent nature is rare. The paralysis is of gradual evolution, and is accompanied by atrophy. Electrically we find that the galvanic reaction fails soon after that of faradism. It should be remembered that the two diseases have been known to co-exist. In a doubtful case, a

return of the fever, and an extension of the paralysis, point to multiple neuritis.

Cervical Pachymeningitis. Multiple neuritis affecting the upper extremities much more profoundly than the lower, might lead one to overlook the fact that they are implicated, and the question might arise whether or not it was a case of cervical pachymeningitis. The possibility of this disease might cloud the diagnosis, since it attacks the nerve-roots, and may produce a paralysis, wasting, and anesthesia affecting the upper limbs and the trunk, but not the lower limbs. There is no tenderness of the nerve-trunks, and there is distinct evidence of disease of the spinal cord, shown by root-pains in definite areas.

Myelitis is a most commonly confused condition. When this disease affects the posterior structures of the cord, so as to give rise to ataxic symptoms, it reminds one of neuro-tabes, or pseudo-tabes, but there are always some symptoms which can come only from some lesion of the cord itself. Complete loss of power in a day or two is strong presumptive evidence of myelitis. The paralysis extends from legs to trunk, and perhaps next to arms. In contrast to multiple neuritis, the pain of myelitis is in the spine, and the paralysis is of the legs (a paraplegia), or of the legs and trunk, and there is rigidity of the muscles, while in multiple neuritis there is flaccidity. There is no electrical reaction of degeneration, except in those muscles innervated from the segments actually implicated in the disease. The deep reflexes are exalted, unless their centers are destroyed, and then they are absolutely lost. The muscles of the back are always paralyzed to some extent, and this loss is in the form of a girdle. The sphincters are always affected, and the atrophic process is so localized, and so acute, that bedsores are common. There is heat in the spine, and if there are anesthetics and paresthesias, they occur in areas like stripes and girdles, and are on the trunk, and affect the upper parts of the limbs before the lower.

In multiple neuritis there is no pain in the spine or trunk, except in a general extension, late, in a very severe case. The paralysis tends to be in some degree simultaneous in the four extremities; never from arms to legs, or legs to arms with an intermediate involvement of the trunk as in myelitis. The muscles quickly show the reaction of degeneration, the deep reflexes are very rarely exaggerated, and then only at the outset, but tend quickly to fade to complete, or almost complete extinction. The muscles of the trunk suffer only in the latter stages of the most extreme cases, and never in a girdle-like distribution. The sphincters are never permanently involved, and the trophic changes are never severe enough to produce bedsores. The tenderness is along nerve-trunks, and in muscles, and about joints, and never extends above the knees.

Landray's Paralysis has been said to present a confusing picture, but the diseases have few points of actual similarity. It has no sensory symptoms, except occasionally some initial numbness and tingling, which is in strong contrast to the severe pains and the exquisite tenderness of the nerves in multiple neuritis. (A case of my own started with tingling in lips, and weakness in legs.) Both have paralysis, but while that of Landray begins in the legs, and moves steadily upward, involving next the trunk, then the arms, and lastly the face, that of multiple neuritis may begin either in the arms, or the legs, and goes to the other extremities, never affecting the trunk, except in those rare and severe cases which are so marked as to permit of no error in diagnosis. The sphincters are rubably exempt in both disease, but while this rule is occasionally infringed by Landray, it is never permanently by multiple neuritis. The course and termination also are strikingly dissimilar. Landray paralyzes the whole muscular system in a few days, and with few exceptions is fatal in from three days to two weeks, by paralyzing respiration and cardiac action, while multiple neuritis consumes from ten days to two months to produce total disability in the parts affected, and tends almost certainly to recovery. Fever to about the same degree may accompany both diseases. The only exception to the gradual onset is in those very severe cases of alcoholic intoxication where the legs may be totally disabled in a few hours to a day or two, but even here the distribution still conforms to the rule as stated, for the trunk is either not involved at all, or not until a much later period.

Hysteria may co-exist with multiple neuritis, and these diseases have been confused when in pure type, although there are essential and ascertainable points of difference. Both present paralysis, anesthetics, hyperesthesias, atrophies, and changed reflexes, but with a difference. The paralysis of hysteria is of a section of the body as thought of in the mind, as of a leg, or both legs; or of an arm, or of one side of the body, or of the whole body; whereas the paralysis of multiple neuritis is of such groups of muscles as are innervated by certain specific nerves. Moreover the paralysis of hysteria has never been a loss of the power of extension, while that of multiple neuritis is peculiarly and distinctively of this character. The anesthetics of hysteria are bounded by straight lines, at right angles to one another, and sharply defined at the borders, and such areas are scattered about the body practically without exception, having at the same time some such areas upon the trunk. There is always some anesthesia of the special senses. In multiple neuritis on the contrary, the anesthetics avoid the trunk, while on the limbs the areas follow the distribution of certain nerves, and the borders are very poorly defined, since the sensory nerves of the skin infringe to some extent upon the borders of each other's territory. In the general distribution of these

anesthesias upon the extremities, both are glove or stocking-like. While the hysterical patient complains bitterly of hypersensitive areas, the calm expression of the face during pressure upon such areas, if the patient be off his guard, negatives the truth of his statement, nor are the nerve-trunks especially tender, nor are they ever swollen. The atrophies of hysteria are slight, and result only from disuse of the part, and never show the reaction of degeneration, while those of multiple neuritis are the contrary in every particular. Hysteria shows no atrophic changes in the skin, while they are frequent in multiple neuritis, the peculiar skin changes of hysteria being insensitive areas which do not bleed upon puncture. These, together with the narrowing of the fields of vision, and a reversal of the area in which various colors can be perceived, constitute what are known as the Stigmata of hysteria, and are not duplicated by the symptoms of multiple neuritis. The deep reflexes are exaggerated in hysteria, and after a primary exaggeration, which is not usual, are diminished almost or clear to extinction in multiple neuritis. Hysteria attacks young people, mostly women, who have been exposed to some emotional strain, and are of a neuropathic constitution, while multiple neuritis affects persons who are somewhat older, and who have been exposed to some intoxication, or who are the victims of some cachexia.

Neuralgia can be diagnosed from multiple neuritis from the fact that it presents more spontaneous pain, and also that this pain is liable to exacerbations at certain fixed times of the twenty-four hours. It is almost always unilateral, as opposed to the symmetrical distribution of multiple neuritis, and while there will be areas of exquisite sensitiveness for a short distance along the nerve-trunks, this varies greatly during different periods of the same day, while that of multiple neuritis is of the same grade, while it continues as a symptom, and extends along the whole nerve, or for long stretches. The nerves in neuralgia do not develop the reaction of degeneration. It must be remembered that multiple neuritis may sometimes appear initially as a neuritis of a single nerve. There is no swelling of the nerve in neuralgia.

Spinal Meningitis. The onset is abrupt; the principal feature is pain, which is always girdle-like, but not to the exclusion of shooting pains, and those radiating down the limbs, since they follow the distribution of nerves, and may go up to the head and neck also. These are the so-called "Root-pains." On account of the same irritation of the roots where they emerge from the cord, we get cramps in muscles, twitching of limbs, opisthotonos (a rigid backward curvature of the spinal column), and while the deep reflexes are at first exaggerated, in the end they are lost. The sphincters are involved. There are all varieties of disturbance of superficial sensation. There is some degree of paralysis, "tache

cerebrale," and Kernig's sign. This latter is the impossibility of complete extension of the leg upon the thigh, when the latter has previously been placed at right angles to the body. The best position is when the patient is lying on the back, or is sitting on the edge of the bed or a chair.

Spinal Hemorrhage. The manner of onset and its rapidity, and the distribution of the subsequent pains are quite diagnostic. In any case the onset is abrupt, and often the patient will suddenly fall with a sense of shock of some kind in the back, and immediately find himself paralyzed in his lower limbs. This is the more impressive and confusing to the sufferer, since his consciousness, and acuteness of intellect remain unimpaired. It is soon marked by pain in the back, which will always show some girdle-like distribution, and in addition will radiate over other parts of the body. The sphincters are involved, and the deep reflexes will be changed, either increased, or lost, according to the site of the lesion. The cranial nerves are spared.

TREATMENT. When the pains appear, warm bathing, and gentle stroking, must be supplemented by hot fomentations. (Osler and others advise hot lead-water and laudanum.) The best method is to apply warm fomentations for half an hour, and then replace them by cotton compresses for four hours. Mills has advised alternate hot and cold sponging, but in all cases the low vitality of the nerve must be kept in mind, and too energetic measures are harmful. If the patient's heart is not weak, vapor or Turkish baths are of value. As soon as the patient can bear the application, a very mild galvanic brushing of the surface will help the pains to some extent. All this period should be passed in surroundings insuring the greatest amount of air and sunlight. When contractures develop in the slightest degree, they should be vigorously combatted by posture in the first place. The bed-clothing should be so arranged as not to weigh upon the toes, which tend to drop, the feet and legs should be placed in normal positions, and held there by pillows or sandbags, or by splints of plaster which are removable, so as not to forego the daily bath and friction. Passive motion of all joints in full extension should be practiced daily. Strapping may sometimes be found useful. The wrist is to be kept on a splint, if there is danger of contracture. If the muscles of deglutition are impaired, feeding should be by stomach or nasal tube, and rectally if necessary, since nutrition by some route is absolutely essential, and should be simple, nourishing, and sufficient. As soon as repair begins, the muscles must be daily exercised by means of the galvanic current, always using large electrodes, and avoiding fatigue. The first response will be obtained by means of static sparks. As soon as the faradic response appears, it should be the current used, and each muscle should be given two or three vigorous contractions daily. Swedish

movements against resistance are useful. Atrophy and degeneration can best be antagonized by persistent massage. If after the attack, the toes tend to bend under the foot, and so impede walking, it is best to put them in a corset-shoe, and build up the heels. A rubber muscle will aid in another way. The patient should not get up until pain and tenderness have subsided, and there is evidence that the process of repair has become established.

Removal of the cause is the first measure to be thought of: removing a person from a poisonous occupation, or preventing the absorption of more of the poison from the water which they drink, or the air which they breathe. It has been found that even old drunkards bear abstinence well under these circumstances, if they are well-fed, and kept warm. Lead has been diagnostically stated to be easily eliminated by the administration of Iodide of Potash. In this procedure, however, it has been asserted that there was a great danger of flooding the body with a soluble iodide, by which a more severe and widespread poisoning was produced. In view of this, T. A. W. Ogg advises that the use of Iodide of Potassium should be accompanied by the free drinking of Sulphur water, by which the soluble iodide of lead is changed into an insoluble sulphide, and so passes harmlessly out of the system. Professor Dixon Mann, however, states that elimination is a very gradual process, uninfluenced by any of these means, but is slightly accelerated by warm baths. There is no particular antidote for Mercury. Antitoxic serum in diphtheritic cases. Comby reports a child of four years cured in three days of a severe paralysis. An old case, which had resisted other treatment, in a short time responded. Thinks all cases should get it, as the virus may not have exhausted its virulence. Others deny its efficiency, and consider it absolutely bad.

In the acute stage, rest and warmth for the affected parts is about all that is efficient, and this is best assured by keeping the patient in bed, with the affected portions of the body swathed in cotton, and wrapped with a flannel roller. If a case is very severe, a water bed may be needed on account of the tenderness of the muscles. It not only abates the pain, but supports one if very weak, and also lessens the danger from a debilitated heart, and impaired respiratory muscles. From an early date, a daily warm bath and gentle friction allay pain, and reduce the amount of muscular degeneration.

Electrical Treatment. When the case has failed to respond to such measures, and we find that the muscles are unresponsive, or have been neglected, and contractures and deformities are present, we must wake up the muscles at first with static sparks, and then follow with galvanism. Contractures must be broken up, if necessary under an anesthetic, and fixed by a splint or dressing. Faradism is best for cutaneous anesthetics, and chronic wrist-drop should be corrected by apparatus.

Electricity cures rapidly in those cases where reactions have not changed to the point that faradic irritability is lost, and in these cases the faradic current is preferable. When faradic irritability is lost, galvanism may be used. When treating a case with the faradic current the high-tension coil should be used, and the amount of current should be small, not enough to produce contraction. The application of one pole should be made to the affected nerve, while the other is over that section of the spine which gives rise to the nerve. In using the galvanic current, treatment should be given labile with the anode over the affected nerves, and the muscles affected by them, and the cathode resting over that section of the spine which gives rise to them. The special point in the treatment of this disease is to use a very weak current, five to ten milliamperes, but continued a very long time, at least twenty-five or thirty minutes. This treatment has proved far more successful than the administration of stronger currents for a shorter space of time. Static electricity as a routine treatment does not give as beneficial results as galvanism and faradism, and, if employed, the static spray administered for some time, twenty to twenty-five minutes, gives the best results, and is most beneficial in those cases where sensory changes are present. Cohn recommends labile combined galvano-faradic treatment of the affected parts. Where irritability has ceased all electro-therapeutic measures are useless.

Vibration is a good remedy for the pains. Place the hammer over the painful point, and treat until relieved, and over the solar plexus, and nerve roots on each side of the spine.

ALCOHOLIC NEURITIS. Electrical treatment greatly hastens recovery, showing rapid improvement, after the first or second treatment. The only contra-indication is severe and persistent pain. If there is no reaction of degeneration present, the faradic current may be used, but even in this case the galvanic is better, and when the reaction of degeneration is present the galvanic should always be used. The treatment is the same as in multiple neuritis. Jones speaks highly of the sinusoidal current for this disease, especially when given through the medium of the bath.

THERAPEUTICS. Our literature is full of remedies, but the most accurate reports seem to indicate that our results are just as dependent as in Physiological medication upon finding and combating the cause. The most success comes from ascertaining the cause, and treating the patient as we would if the disease had different manifestations. If, therefore, the case is rheumatic, we should at once think of Bryonia, Rhus, Cimicifuga, or Apis. Symptoms might occasionally turn us to one or other of the more unusual drugs, but if we keep in mind not only the existence, but also the *comparative* value of symptoms, we shall be likely to keep

pretty closely to the drugs mentioned. In alcoholic cases the usual prescription will be either *Nux Vomica*, or *Cimicifuga*. Cases which develop marked tremor would seem to call for *Argentum nit.* conspicuously, among others, but it has not seemed so valuable as in some other diseases marked by this symptom.

Aconite. In the earliest stages, but never if tingling is not a marked symptom.

Argentum nit. Post-diphtheritic, marked by dilatation of the pupils.

Arsenicum. This is especially indicated in lead palsies. The pains relieved by this drug are worse at night, are better by heat, and there is marked mental restlessness.

Apis. Shows numbness with burning pain, and great sensitiveness.

Agaricus. The presence of much muscular jerking, and twitching.

Arnica. Where there is not so much pain as soreness, the patient shrinks from being even approached, and there is aggravation from motion.

Belladonna. When high fever marks the attack.

Bovista. For the stage of numbness and tingling.

Cocculus. Tingling of limbs with paralytic trembling.

Colocynth. In infrequent cases.

Calcarea carb. For the chronic conditions.

Carboneum sul. In same. Especially suited for cases affecting the territory of the median and radial nerves. Pains and jerkings, particularly at night.

Ferrum phos. Dead feelings in the hands, and a soreness of the muscles upon motion.

Gelsemium. Paralysis with marked tremor. Markedly motor.

Hypericum. Is characterized by as great fear of approach as *Arnica*, but it has more sensitiveness, and the spine is especially sore to touch. It is more often a neuralgic remedy than one for neuritis.

Lathyrus, and *Ustilago maidis*. Have produced the lesions in nerves, but the clinical results have been disappointing.

Magnesium phos. Numbness of hands, with lightning pains running up the limbs.

Nitric acid. Is called for by a lancinating pain, but it is more often neuralgic.

Physostigma ven. Is especially indicated for ocular palsies from diphtheria.

Phosphorus. Has been highly endorsed.

Plumbum. For diphtheritic cases marked by extreme hyperesthesia.

Secale corn. Is in the class with *Lathyrus*, and *Ustilago*. Cases with paresthesias.

Silicea. Has good clinical reports.

Zinc. Is highly spoken of, but has never seemed to favorably affect an organic condition.

In the stage of fever the physiological treatment has been antipyretics of all kinds. In alcoholic cases Citrate of Potash, Nitrous Ether, Tincture of Cinchona, and Digitalis if the heart is weak. In rheumatic cases the Salicylate of Soda, but if the patient be also alcoholic the Salicylate of Potash should be substituted to avoid the combination with, and retention of, the uric acid by the Soda. No drug is specific, and while Mercury is more efficient in the interstitial varieties of neuritis, it still should be tried in polyneuritis when the nerve-sheaths seem to have become affected. Antipyrine and Mercury in small and repeated doses are of great value in rheumatic cases. Mills advises Gaultheria for rheumatic cases. In toxemias like septicemia give Tr. Perchloride of Iron in doses of 20 to 30 drops t.i.d. Mercury and Kali Iodide for syphilitic cases. After the first shock is over Iron, and Strychnine in small doses, and Quinine. Others say Strychnine in increasing doses.

Iodide of Potash is used extensively, but for some unknown reason is good only for the sensory forms. Arsenic, in small doses also, in the sensory cases only. It is advisable to add one-twelfth to one-quarter grain of Cocaine to whatever other remedy is given if the patient be an alcoholic, as it stops the craving, and renders the stomach more tolerant of food.

Quinine very freely for the malarial form. This form can only be differentiated at times by the action of this remedy.

The use of anodynes is always likely to become important, and the Coal-tar series is usually better than the opiates. Cocaine hypodermatically one-fourth of a grain seems to aid regeneration, and does not simply curb the pain. Sleep is most certainly produced by a mixture of Bromide and Chloral, but Trional, Sulphonal, Chloralamid, Phenalgin, and Antipyrine, are useful in about the order given.

Mental excitement is most surely antidoted by Hyoscine 1-400th to 1-150th of a grain.

After the febrile state use a general tonic treatment of Strychnia, Iron, Quinine, Arsenic in small doses, as it may increase the trouble. Codeine and Cocaine are better as anodynes than Opium, as the habit is less easily formed. In lead cases purge freely, and give Opium in the colic stage.

LANDRAY'S PARALYSIS.

DEFINITION. It is an acute parenchymatous degeneration of the peripheral motor neurons, of toxic or infectious origin. This process gives rise to an acute ascending motor paralysis. It

begins in the lower extremities, and rapidly extends upward, involving successively the feet, legs, trunk, arms and face, and the muscles of the diaphragm, heart and pharynx.

THE SYMPTOMS OF A TYPICAL CASE. There is rapid paralysis of the whole body occupying only a few days in its development. It progresses from below upward, until it involves the face and the muscles innervated from the bulb. The sphincters are not disturbed, there are no sensory disturbances, and the mind remains clear.

DIFFERENTIAL DIAGNOSIS. This is from Meningeal Hemorrhage, Peripheral Neuritis, Anterior Poliomyelitis, and Myelitis.

AGE. It is a disease almost exclusively of persons between the ages of twenty and forty. It has been noted in children.

SEX. With few exceptions it is confined to the male sex.

ETIOLOGY. It commonly occurs in the colder months and refrigeration seems competent to be the sole cause in some cases. The author observed a case which was solely attributable to exposure combined with over-exertion, during the blizzard of 1888. Another case occurred when, after a debauch, the "Keeley Cure" was invoked for the third time. It has seemed to result directly from alcoholism, and much more often from injuries received while under the influence of alcohol. It has followed the infections during convalescence. It has succeeded traumatism, and infections, marked by the formation of foci of pus. Cases occurring in the puerperium are doubtless due to such a cause.

PATHOLOGY. It was formerly considered (so stated by Landray in 1859) to be without an assignable lesion, but later investigation has uniformly revealed lesions, but not of definite location. The study of one group of seven cases led the observers to conclude that it was a peculiar manifestation of peripheral neuritis, but the latest conclusion seems to be this. It is a disease which may occur with any one of four groups of lesions. First: there may be changes in the peripheral nerves which indicate interstitial and parenchymatous neuritis. Second: there may be changes in the spinal nerve-roots only, particularly in the anterior ones. Third: there may be disseminated foci of inflammation, exudation and capillary hemorrhage in the medulla and cord, and swelling of the antero-lateral tracts, and in the anterior horns. Fourth: there may be changes in all parts of the lower motor neurons, peripheral nerves, spinal roots and ganglion-cells due to a scattered and diffuse pathological process. It is an acute parenchymatous degeneration of the peripheral motor neurons, of toxic or infectious origin.

SYMPTOMATOLOGY. Generally there are no warning symptoms, but in one of my own cases there was a feeling of malaise with a numbness of the lips for about twenty-four hours previous to the advent of any notable paralysis. Others have noted tingling in

other parts as a premonition. The first diagnostic symptom is a weakness in the lower extremities, which becomes a relatively complete paralysis in from twenty-four to forty-eight hours. The weakness and paralysis are not invariably in the lower limbs, but may be in the gluteal region or thighs. Extension of the process then ensues, and is steady and rapid. If the parts other than the lower legs are first affected, the first sign of extension will be their involvement, but, in any case, after the legs are paralyzed, the trunk is next affected, and as the paralysis approaches the thorax the respiration becomes weakened, and may be the cause of immediate death. Next, the arms become weak, and finally are as paralytic as the legs, but before they are greatly affected, the nuclei in the bulb betray the inroads of the disease, and speech, and deglutition, but most vitally the action of the heart, are involved. Death is most commonly from this latter effect, and ensues so soon as these centers are affected. There may be facial and ocular palsies in slow and mild cases. The deep-reflexes are abolished as the disease reaches their centers, excepting only in those cases which are so slow in their progress that there is an interval of irritation of their centers before their ultimate destruction. Since the lesion may be a diffuse one, we may find loss in one part and exaltation in another, but loss is the final fate of all. The sphincters are not disturbed, but, owing to motor weakness we may find a simulated retention. While the muscles lose their tone, they do not show any actual atrophy, except in those very slow cases whose rate of progress casts doubt upon the correctness of the diagnosis. During all this paralytic process sensation is undisturbed, except that once in a while a slight dulling of perception may be found, or a little pain in the affected muscles, in the latter case when the process has been a very rapid one. Temperature is sometimes elevated, albumen found in the urine, and some enlargement of the spleen; all these symptoms are confined to the infectious cases. Electric changes are absent. There are no mental symptoms.

COURSE. Paralysis of the lower limbs is generally complete in one or two days, and of the whole body in from three to four days, although doubtful cases are reported which have run a course as long as four weeks. Remissions are possible at any stage.

PROGNOSIS. Almost always fatal in from three to four days to a week, but a small number of cases have recovered. Spiller quotes one. The hopeful condition is one where there is a stationary condition of stasis for a few weeks, and then a slow recovery of power.

DIAGNOSIS. Cases generally present no difficulty, since no other form of paralysis presents such a combination of rapidity of onset, progression without skipping of parts, absence of sensory symptoms, sphincter disturbance and bedsores.

DIFFERENTIAL DIAGNOSIS. Meningeal Hemorrhage has an even greater degree of suddenness, but it has pain in the back (sensory symptom), there are spasms of the muscles, and exaltation of the deep reflexes for a short time in the majority of cases, and finally, the sphincters are surely disturbed to some extent, and if the hemorrhage is a large one, there will be bedsores and atrophy of muscles.

Peripheral Neuritis is very like some cases of the slower kind. The sudden neuritis from alcohol or diphtheria is distinguished by the presence of sensory symptoms, in the case of alcohol, and by the primary pharyngeal, or respiratory, or ocular site of invasion of diphtheria. In all forms of neuritis the history will generally be a sufficient guide; if not, the superior rapidity of progress of Landray's paralysis, and the fact that it involves the trunk before the arms, should correct the error.

Anterior Poliomyelitis paralyzes a limb, or one side, or is paralytic in a few hours after a sudden fever (this fever may in some cases be too transient and slight to attract attention), and in a day or two regresses, so that only a part of a limb is paralyzed. This paralysis soon becomes atrophic. The paralysis is rather wide in extent, and speedily grows less in extent, but is degenerative, while in Landray's paralysis it is limited at first, continually extends, and is not degenerative.

Transverse Myelitis affects a limited area which can be marked out by very definite sensory symptoms (root-symptoms), while Landray's paralysis has none. It is degenerative, producing bedsores, and affects the sphincters, which are also points of difference.

TREATMENT. The patient should be kept absolutely at rest, and the limbs should be kept warm to keep up the most efficient circulation, either by wrappings of flannel, or the continuous warm bath. There is no objection to moderate massage, but while electricity may be used, employing mild currents, it has no very great promise. If a case tends to recover, faradism should be of value in the convalescent period. There is general agreement that some method of stimulation of the spinal circulation is theoretically valuable, and for this purpose friction, the negative galvanic current, irritation by sinapisms (mustard one part and flour three parts), and even the actual cautery have been advised. In one case which recovered, the whole spine had been kept constantly irritated by a sinapism. In all cases nutrition should be abundant and stimulating, and pure stimulants should form a feature of the treatment. If deglutition is affected, the nasal tube should be used, and in cases of paralysis of the glottis tracheotomy should be made. Injections of ergotin were used in another case which recovered.

THERAPEUTICS. The drugs which have been recommended for use in this disease are of doubtful efficiency, since almost all of

them are conspicuous by the abundance of sensory symptoms found in their pathogenesis, while this disease totally lacks them in some cases, and in none of them is there more than some slight parasthesia. The recommendation that it should be treated as a myelitis is not sound, since neither the pathological findings, nor the symptoms resemble that disease. In one case of the author's the administration of Oxalic acid in the sixth potency was followed by an amelioration of the symptoms, but cardiac failure and death followed the apparent improvement. Aconite may be of use where the paresthesias are prominent in the form of tingling numbnesses. Conium maculatum has a better basis, since poisoning by it produces a paralysis spreading from below upward, and finally causing death by failure of respiration and cardiac action, all with an unclouded sensorium. Hydrophobium; after inoculation with the serum of Pasteur some cases have died with the symptoms of Landray's Paralysis. In at least one of these cases the symptoms were so similar that post-mortem examination was necessary to correct the diagnosis. The following remedies have been advised: Alumina met, Lathyrus, Ledum. Phosphorus, Rhus tox, Secale.

CHAPTER III

DISEASES OF THE SPINAL CORD

System Diseases

TABES DORSALIS.

LOCOMOTOR ATAXIA—POSTERIOR SPINAL SCLEROSIS.

DEFINITION. This is a chronic degenerative disease of the nervous system, affecting specifically, and most constantly, the sensory nerves and sensory tracts in the cord, and from such degeneration arise the characteristic ataxia, and sensory derangements. While there may be an extension of the process, any motor involvement is accessory and unessential, the sole essentials being muscular incoördination and loss of knee-jerk. It is not a paralysis, but an incoördination. It is generally, though slowly, progressive, and the patient finally becomes a hopeless invalid. Among nervous diseases it is a common one.

THE SYMPTOMS OF A TYPICAL CASE. A person over forty years of age begins to suffer from double vision, from lightning pains in the extremities, and from incontinence of the bladder. He is sensible that he feels compressed as by a girdle about the body, or one of the limbs. He finds that he cannot stand steadily without the aid of vision, and that he has lost impressions of touch in his feet, and perhaps also in his hands. On examination it will be found that he has lost the deep reflexes, that he has insensitive areas upon the skin, and that his pupils will react to accommodation, but will not to light.

DIFFERENTIAL DIAGNOSIS. It is to be especially distinguished from Multiple Neuritis, General Paresis, Syphilitic Pseudo-Tabes, Spinal Syphilis, Multiple Sclerosis, and Hysteria, all of which present marked similarities, and may co-exist. It is less often similar to Hypochondria, Exophthalmic Goitre (of an aberrant form), Neurasthenia, Chronic Myelitis, Spinal Tumor, and Hereditary Ataxia. Multiple Sclerosis, Ataxic Paraplegia, Cerebellar Tumor, and Syringomyelia may at times resemble it.

AGE. A disease of the fourth to the fifth decade of life, old age and youth being practically immune. The juvenile cases are practically always expressions of hereditary syphilis.

HEREDITY. It is doubtful whether it is ever actually transmitted from parent to child, but a sufficient number of cases have been reported of several persons in the same family being affected, to raise a suspicion of its possibility.

ETIOLOGY. In literature tabes has been ascribed to a large variety of causes, of which syphilis is only one. Fright, over-exertion, refrigeration, have been considered possible causes. Present opinion, based upon carefully collated reports, has given to syphilis a proven percentage of from 60 to 90 per cent. Gowers says that "Syphilis is a '*conditio sine qua non*' of the occurrence of tabes." Erb says that of 100 cases in his own practice, 90 per



FIGURE 49.—TABES DORSALIS.

Site of degeneration is shown in the right half as a white area. By referring to the left (schematic) half it will be seen to have wiped out the tracts of Goll (7), and Hurdach (8), and to have affected the fibres, and secondarily the cells, to some extent, of Clarke's column (18).

cent have had syphilis, or a chancre, and of the remaining 10 per cent. only 3 per cent. were free from a suspicion of it. In regard to the status of this 3 per cent. a statement of Pernet is interesting. He found that in the dermatological clinic of the University Hospital in London he could *prove* a previous syphilis in only 80 per cent. of those presenting undoubtedly syphilitic lesions of the skin. The causative agent is, however, not the syphilitic virus itself, but a later toxin which has been called parasyphilitic. It has been suggested that tabes is the result of a syphilitic toxin plus x —(x being an unknown and intensifying element). Edinger considers that, among others, tabes is more of an exhaustion disease than a toxic one; that is, there is a poison plus a functional over-

use of a part, which combination produces a using-up disease, or *Aufbrauchkrankheit*.

PATHOLOGY. Ferrier, at the Lisbon Congress of 1906, said: "The current theories are these—There is a theory of interstitial sclerosis following an endarteritis; Redlich and Obersteiner's theory of lepto-meningitis; Marie's theory, according to which tabes is the result of a syphilis of the lymphatic system of the spinal membrane; the theory of a neuritis following a local meningitis; and, finally, the theory of Thomas and Hauser, according to which tabes is to be regarded as a dystrophy affecting principally the central neuron. Ferrier regarded this last view as the proper one. An examination of the cord of a tabetic will show that the fibres coming into the posterior portion of the cord are the most common seat of degeneration, and this may be so complete that in the cauda equina the empty neurolemma sheaths may be all that remain of the former bundles of nerve fibres. Owing to the fact that these posterior columns do not wholly consist of fibres coming from without, but are partly made up of fibres originating from within, and associative in function, the degeneration of the posterior areas of the cord is not a complete one. In its most discreet form it is a degeneration of a crescent-shaped area lying to the inside of each posterior horn. These areas are termed *Rubans Externes*, or *Bandelettes Externes*, by Pierrot, who isolated them in 1871. In these *bandelettes* run three bundles of fibres. The first group is composed of short fibres, which lie in the middle of the *bandelettes*, and are distributed to the cells of the posterior horns; the second group is divided into two portions, one of which goes to the cells of the anterior horns, and the other to the cells of the column of Clarke (sensitivo-reflex group of Kolliker). This group lies in the anterior portion of the *bandelette*, and the fibres are of median length. The third group lies on the posterior aspect of the *bandelette*, and is composed of long fibres, which, entering the cord near the posterior horn, continually tend toward the center line, and under the name of the column of Goll go the whole length of the cord, and end at the medulla in the nucleus gracilis. All these long fibres are from the lumbo-sacral and lower dorsal segments. The tabetic degeneration, so evident in the posterior columns, seems to stop short at the spinal ganglia, and the sensory nerves distal to the ganglion are normal, but not infrequently the final peripheral ramifications in the skin, muscles, and other tissues, undergo an atrophy similar to that found in the cord. Batten found that certain end-organs of the sensory nerves in the muscles, the so-called "muscle-spindles," were uniformly diseased in all those diseases which were marked by ataxia, and were very much less degenerated where this symptom was only slightly evident. The fibres affected by such a degeneration would include those ending about the cells

of the column of Clarke, and it is therefore evident why these cells and fibres are the portions of the posterior column of the cord which are earliest and most constantly degenerated. The posterior root ganglia show hemolysis, pigmentation, fatty changes, occasional deformity of, or diminution in the number of cells, and an increase in the connective tissue elements. These results are similar to those resulting from the experimental division of the roots in animals, but it is doubtful if the amount of change discovered is sufficient to account for the extensive degeneration of the terminals of the nerves, and of the posterior roots themselves. It is rather such a degeneration as has been termed retrograde, in which case the injury has been first to the fibres, and when the cell condition is a result of such injury, and not the cause of it. While constantly and most distinctively there is a change in the posterior elements of the cord, degeneration is not always confined to this area, but is also displayed in the optic and sympathetic nuclei, and certain motor neurons.

SYMPTOMATOLOGY. As a general truth, it may be said that from ten to fifteen years after an infection from syphilis, the patient will find himself suffering from certain disabilities for which he will seek advice from a physician. Among initial symptoms there is a wide possibility as to which will first attract his attention, and investigation has frequently revealed that certain deviations from health had long existed, which he had only casually noticed, and had endured without having investigated their meaning, or which had been so transient that they had passed out of his mind. The tables which give percentages of initial symptoms are not correct representations of the particular symptom which first drove the patient to a physician, but are based upon the results of inquiry. As a rule, the patient is most liable to complain first of darting pains in the legs, or that he cannot hold his urine, or that he has a great deal of vertigo, and, once in a while, that he has become so ataxic that he cannot go about a room in the dark, or maintain his position while he bends over the bowl to wash his face in the morning. A patient of my own first discovered that he was ataxic from getting into trouble frequently, on account of bumping into people upon the street. Sometimes a recurring causeless abdominal colic will attract attention, or it may be the presence of double vision. With these pains, or with most of the other symptoms mentioned, he may have little conscious ataxia, and so he should be placed in the Pre-ataxic stage, which may last from a few months to ten or more years. Even at this time, however, a careful examination will disclose a diminished, and probably a lost knee-jerk, an Argyll-Robertson pupil, and there will be in some parts of the body an inability to correctly appreciate touch and pain. If it is found that he will stagger if induced to stand erect with the eyes closed, and the feet closely apposed

(Romberg's sign), he has already entered upon the second, or Ataxic stage. It will now be found that he cannot execute movements accurately with the eyes closed, nor can he walk well without the aid of sight. By this time there are very definite losses to all sorts of sensations of the skin, his bladder and rectum are under poor control, he is tormented by pains in the limbs, and pressure symptoms about the body, and his vision is probably much impaired. This stage may last from two to ten years, with long halts, and some remissions to his suffering, but soon there will be an increasing weakness, the appearance of atrophies about the joints, and when they have reached their full developement he has entered upon the third, or so-called Paralytic stage. This is hardly ever a true paralysis, since he generally has the power to execute any required movement, (unless the joint disintegration has reached a high grade), but his ataxia is so great that his power is misdirected. His mind now tends to degenerate in the direction of a mental feebleness, rather than into any actual or definite psychosis. In order to more specifically describe these changes, they will be taken up as they are classed in the following table (from Church and Peterson), in the order of their rutable sequence:

1st. Pre-ataxic stage. 6 mos. to 20 yrs.	2nd. Ataxic Stage. 2 to 10 yrs.	3rd. Paralytic stage. 2 to 10 yrs
Motor. Eye-palsies. Ataxia. Muscular weakness.	Less. Increased. Paresis.	Increased. Paraplegia.
Sensory. Pains.	Pains. Anesthesia.	Pains less. Increased.
Reflexes. Knee-jerk lost. Argyll-Robertson pupil.	do. do.	do. do.
Trophic. Arthropathies.	More rare.	Rare.
Visceral disturbance. Sexual weakness. Vesical weakness. Constipation.	Increased. do. do.	Increased. do. do.
Special Senses. Diplopia. Optic atrophy.	Rare. do. Deafness.	Increased. do. Paralysis of accommodation.

Eye Palsies. All the muscles of the eye may be paralyzed, but the ciliary and trochlear almost always escape. Usually we find ptosis (drooping of the upper lid), and squint (strabismus) usually fleeting at first, lasting from a few days to a week, but finally becoming permanent, and the squint produces diplopia, (double vision). It may not be apparent to the patient, and be first discovered on examination of the eye for other losses. A ready test is made by covering one eye, warning the patient to keep both open, and having the patient follow with the uncovered eye the movements of the examiner's finger. This should be moved in all directions, finally ending in causing extreme outward rotation of the eye. Now quickly uncover the other eye, and observe the relative position of the two eyes. They should preserve their parallelism, in spite of the fact that one has been covered. Examine next, ending in extreme inward rotation. It is diagnostic that it is from tabes when it is not relieved by specific medication, for usually the only other probable cause is syphilis.

Ataxia. Under the above heading we should class several defects, which are often viewed as separate conditions, thereby obscuring their common origin, namely, Hypotonia, Incoördination, Static Ataxia, and Asteroignosis. These are manifestations of a common loss, namely, the lack of appreciation of pressure and tension on skin, tendons, muscles, and joint surfaces.

Hypotonia. This is the loss of the power of smoothly executing movements with the weighty parts of the body. The patient betrays it by dropping into a chair, instead of gradually seating himself; or, more accurately, if the patient, lying on his back with the knees bent, and touching one another, and with covered eyes, is asked to permit the legs to smoothly and slowly fall apart. He cannot accurately estimate the relations between muscles and their antagonists, and therefore executes the movement in a jerky hitchy manner. It is said by some to be absolutely the initial symptom.

Incoördination. This is essentially a sudden and immoderate action of the prime movers of the body, on account of a lack of appreciation of the proper amount of resistance in the antagonistic muscles, since no movement can be smoothly executed without a nice adjustment of the two sets of muscles. A simple test for this loss consists in the examiner touching the heel of one leg upon the patella of the other. The patient is then asked to repeat the manoeuvre, with closed eyes. He will then grope about with the heel, thus making manifest the degree of incoördination. If the tabes affects also the upper portion of the spine, the patient may be asked, with closed eyes, to touch the tip of the nose with the tip of the first finger, the arm having previously been extended to its full length out at the side of the body. The handwriting will also show this same jerky incoördinate movement. Such

movements can be very accurately executed by a person in health.

Static Ataxia. Said to be the first symptom in 3% of the cases, but may exist undetected for a long period, since it is not painful, nor very obtrusive. It consists in an inability to walk or stand steadily without the aid of vision. It is revealed by Romberg's test (described above), and depends upon the presence of anesthesia of the soles of the feet (a similar loss occurs, at times, in the hands), and the loss of appreciation of tension in muscles, joints, and tendons. It naturally is an early result of these losses, since both standing and walking depend upon a constant and delicate readjustment of muscular effort, and the tabetic has lost many of the nervous end-organs and tracts which formerly furnished him with this information. Static ataxia rubably increases as the disease progresses, but (with scanty evidence) it has been asserted that as sight fails static ataxia disappears.

Asteriognosis. In a state of health, we can, within 5%, appreciate the difference in weight of two similar objects held in the hands, without the corrective aid of vision. More than this, we can appreciate slight differences of form and texture, or peculiarities of a surface. The loss of this faculty is not a constant symptom in tabes, and it occurs also in other diseases, since it is perfect only when the receptive centers of the cortex, the sensory conduction paths of the cord, and the receptive peripheral apparatus are in a healthy condition. It depends upon the perfection of the spacing sense, the localizing sense, the muscular sense, the pressure sense, the temperature sense, and the pain sense. This is Steriognosis, and its absence is Asteriognosis.

Muscular Weakness. This is generally an early symptom, for there is always a degree of anemia, and it endures throughout the disease, but in only 5% is there any general paralysis, and whenever it occurs, it is a complicating condition. Separate muscles exhibit normal strength, even in the late stages of the so-called paralytic phase. The helpless invalid will execute simple movements with adequate force, his actual loss being that of incoördination. Of course in the presence of extensive joint-destruction he will necessarily be helpless. The accessory paralyses indicate that the nuclei of innervation, or motor tracts, have been invaded, but such cases are rare, and the results are generally temporary. The most common paralysis is facial, next lingual, then laryngeal. We may find paralyses of a single part (monoplegia), or of the lower limbs (paraplegia), or of half of the body (hemiplegia), and sometimes the patient suffers from a sudden giving-way of the legs, which is a transient form of paraplegia.

Sensory Disturbances. Loss of sense-appreciation is an early symptom, and since we are dealing with a disease of the sensory nervous system it must be present to some extent in all

cases. These losses will be those of which the patient himself is conscious (subjective), and those of which he may or may not be cognizant, but, in any event, can be discovered by another (objective). They may be an exaltation of sensation, or a diminution, and there may be all varieties of impairment of the perception of touch, temperature, or pain. If merely changes occur without more accurate definition, they are called paresthesias; if the sensation of touch is lost, it is anesthesia; if it is diminished it is hypesthesia, while it is hyperesthesia if it is exaggerated. If the perception of pain is lost, the condition is called analgesia, while if it is exaggerated it is hyperalgesia. If the sense of variation of temperature is lost, the condition is thermesthesia. The earliest subjective symptoms are paresthesias, creepings, crawlings, but more commonly there is a numbness and anesthesia of the soles of the feet, the tips of the fingers, and the palms of the hands. A more definite distribution of such losses is rarely appreciated by the patient. Both subjective and objective anesthesia are always to be found in the second stage, and the loss of bone-sensation is said by Jean Heitz to antedate cutaneous changes. It is tested by applying to a bony surface a large tuning fork, giving 128 vibrations a second. These vibrations are perceptible throughout the bone in health, but lost in tabes.

In all but a very small proportion of cases (9 out of 450 in Starr's group) pain was a symptom, and is apt to be an early one. If early, it commonly continues throughout the disease, but may disappear with the appearance of marked ataxia. If pain appears late in the disease it is apt to be mild, and of short duration. At first it is taken to be rheumatic, or neuralgic. Pains are of two varieties: brief and prolonged. The brief pains are of two classes: superficial and deep; the brief superficial pains are usually felt on, or just under the surface, and usually in one spot. They are extremely brief, but recurrent, and from their character have gained the name of lightning pains. They have the remarkable effect of leaving the skin *very* tender, (*Plaques douloureux*), and may occur where the skin has lost its sensibility to pain from external irritants. They most commonly occur in the limbs, especially the lower legs and feet. Next are brief, deep-seated pains; not as a rule very definitely located, but sometimes referred to the joints, usually in the limbs, and not followed by hyperesthesia. They are not so momentary as the superficial pains, but last several seconds. Prolonged pains: these last for days or hours in the same place, most commonly in the trunk, and are usually deep-seated, simulating sciatic neuritis, the only superficial variety being the so-called girdle-pains which are generally about the waist. These pains consist in a sense of distressing tension in muscles and tendons, but they may have a burning character. There is seldom any exciting cause. There is one

form of the disease where pains so dominate the scene as to justify a certain classification. Gowers reports 11 such cases, all in adult males, and with a specific history in 9 of the 11. The pains were severe and neuralgic in character. The extremities of the peripheral nerves are said by some to be the seat of these pains, but others consider them root pains, manifested in the areas to which these roots are distributed. Girdle-pains are like a burning band around the abdomen, or sometimes like a narrower band about the limbs. Another form of this nervous reflex is a sense of pressure, hardly a pain, and of much broader distribution about the upper abdomen, or the chest ("tabetic cuirass"). From the same causes arise spasmodic pains in the viscera, which may exist in many situations, and are termed "crises." The gastric and visceral are the most common, and appear as acute gastralgias and intestinal colics, without adequate cause. There are testicular crises, those of the clitoris, nephritic, hepatic, and Oppenheim speaks of those of the larynx: this latter is a painful sort of croup attack, coming on in an adult, and sometimes threatening suffocation. It may be precipitated by pressure on the hyoid bone, and therefore a tight collar should be avoided by those suffering from this form of crisis. Cardiac crises, simulating angina pectoris, have been described, but must be accepted with extreme caution, and after prolonged observation.

Sensory Losses. On examination one will always find patches, strips, and areas of all shapes, where the patient has lost his sense perception; it may be to all varieties of sensation, but most universally to that of pain, or it may be only delayed in appreciation, or may be interpreted as pressure, or felt in another part of the body from that which is the seat of the infliction of the pain. It is practically always found on some part of the legs, sometimes on the body, and also the face. The deeper parts are not immune, and this explains the absence of pain sometimes noted in cases of fracture. An early symptom, by some urged as diagnostic, is the absence of pain on strong pressure of the tendo-Achillis. The testicles are also analgesic. Thies states that a tabetic woman, giving a history of syphilis, had her eighth confinement without pain. He notes the fact that pregnancy does not seem to influence tabes for the worse. Most diagnostic, in combination with other symptoms, is an anesthesia of the palms of the hands and the soles of the feet, objective like the analgesia, since the patient is often oblivious to a lack which the most cursory examination will plainly reveal.

Reflexes. The deep reflexes are always affected in tabes, and speedily lost, without the previous exaggeration found in some other diseases where loss is also the final result. The typical symptom of tabes is absence of knee-jerk, but the Achilles-jerk, said to be an earlier loss even than the knee-jerk, and the

triceps-jerk are also lost. The value as a symptom of the discovery of lost knee-jerk (Westfal's sign) rests upon the fact that most observers affirm that it is never absent in health. (Starr says that it is in 2%), and never lost in any condition except transiently, unless organic disease is present. It may be temporarily lost in some febrile, and post-febrile conditions, for a time after an epileptic attack, and in acute alcoholism. It should not be considered lost unless the examination has been a very thorough one, since anxiety may mask it, or it may be restrained. The method of examination is as follows: Have the patient cross the leg over its fellow at the knee, and, being sure of the position of the patellar tendon, strike it a sharp blow with the edge of the hand, or a percussion hammer. If the patient is in bed, support the leg under the lower part of the thigh, and proceed as before. If there is no response, place the patient in a sitting posture on the edge of the bed, or better a table, with the legs hanging just free of the edge, and repeat the blow upon the tendon. Failing in these ways do not consider that its absence is certainly established. Now use some method of re-enforcement, all of which are based upon the idea of attracting the patient's attention from the matter in hand. The most common is that of Jendrassik. Place the patient as before, then ask him to hook his hands together, and fix his eyes upon the ceiling. At the word of command he is to attempt to jerk the hands apart by an instantaneous pull. At the moment of command the examiner strikes the tendon as before. If no jerk can be elicited, the reflex may be considered to be absent, but if possible the manoeuvre should be repeated at a subsequent date, lest there may have been some error in technique. Laufenauer's method is also useful. Exceptionally the knee-jerk may persist in tabes; viz., if the lesion in the cord is confined to a very definite and small area, which is either too high, or too low, to involve the reflex arc. Bramwell reports a case where the plantar reflex was exaggerated, and the knee-jerk present; and one where the Achilles-jerk was absent, and the knee-jerk persisted.

Superficial, or Skin, Reflexes. These are not constant in their condition; being exaggerated in some cases; but even if exaggerated at the outset, they are usually diminished or lost in the later stages. In one case an exaggeration of the abdominal reflex antedated all other symptoms by two years, and an exaggeration of the abdominal with loss of the knee-jerk is said to be an early symptom of the disease. The cremasteric reflex (sharp retraction of the testicle when the skin of the inside of the thigh is stroked) is diminished or lost. Tonicity of the sphincter-ani is diminished or lost.

Organic Reflexes. A disturbance of the functions of the rectum and bladder is an early symptom in 80% of the cases,

and arises from the imperfection of the sensory apparatus. The common rectal difficulty is an obstinate constipation, but once in a while involuntary defecation will harass the patient. The bladder fails in function by a loss of the control of the sphincter, or, even earlier, from an anesthesia of the urethra; the patient may wet himself from an inability to appreciate when he has finished micturition. Instead of relaxation there may be retention, or slowness in starting the stream, but catheterization should be deferred, since after some delay, or in some position, the patient is usually able to perform the act. These defects become more pronounced in the later stages of the disease.

Sexual Power is usually lost at an early date, and never returns. It is often the loss which drives the sufferer to a physician, although not an early symptom. It may be preceded by an annoying priapism, if the case is one where the lesion is situated in the upper part of the cord. If this apparent increase in sexual power is gratified it tends to increase the pains and the ataxia. 50% of cases show this loss.

Pupillary Changes. These are actually organic reflexes. These changes are of great diagnostic importance in tabes. It is very significant, and should stimulate one to further investigation, if the pupil is found to be of unequal size in the two eyes, or to differ from the normal by peculiarities in shape or position. The most distinctive pupillary condition in tabes is Argyll-Robertson pupil. This consists in the fact that while the pupil will react normally to accommodation for objects at variable distances, it has lost its power to modify its aperture proportionately to differences in the amount of light to which it is exposed. Remember that it is found in other conditions. It has been seen in diabetes, amyotrophic lateral sclerosis, and poisoning by bisulphide of carbon; it occurs in general paresis, at times in constitutional syphilis, exceptionally in alcoholism, temporarily after railroad accidents, and other shocks followed by no evidences of a grave neurosis. It is an early symptom in 90% of the cases of tabes, but its pathology is doubtful, although it probably depends upon a degeneration of the ciliary ganglion. The reaction to accommodation is better preserved than that to light, since the duration of the stimulus is so much longer that the blunted perceptive power can receive the impress of the one, and fails in the other. Do not feel that the condition is proven if the reaction to light is absent, for the test for accommodation is needed to determine that the eye has not undergone previous changes which would neutralize the test. Test for accommodation by observing the dilatation of the pupil when the eye is fixed upon a object at a distance of ten to twenty feet, and its subsequent contraction when the gaze is fixed upon the examiner's finger, approached within eight to ten inches of the patient's eyes. The difference

in the width of the pupil should be marked. Examine the light reflex thus: face the patient toward a good source of light, and then observe if the position is such that the outline of the pupil can be clearly seen. Some care must be used, since some eyes will reflect confusing images unless the illumination is somewhat lateral. This is peculiarly true of the brown and black eye. Now cover both eyes with the examiner's hands, warning the patient to keep both of them wide open. On uncovering one eye quickly, the pupil should contract sharply. Slowness of response is evidence that it is only a stage of a process that will end in loss. Try both eyes, since the loss at first is occasionally unilateral. If the patient is in bed flash a match, light from a candle or other source of illumination before the eye. Holding the lids closed, and suddenly pushing them up may be sufficient. The investigation may require considerable care, as the pupil of the tabetic is apt to be small, as well as stiff. Be cautious in the case of elderly persons in being assured of the presence of the power of accommodation, since in the absence of this we should not expect the reaction to light.

Optic Atrophy. This may come on at any stage: from 1 to 2% have it as an initial symptom, and from 20 to 35% have it in some stage of the disease. It may antedate all other symptoms by years; it is of the gray variety, generally bilateral, and occasionally it is preceded by choked disc. It never co-exists with strabismus, or ptosis. The patient may be unconscious of his loss for years, since central vision is likely to be preserved for a long time. The loss may be in the width of the field of vision, a section of the field may be cut out, or there may not be a blindness, but only a dimming of the whole field. There may be a loss of one-half of the field (Hemianopsia) and the loss may be bitemporal, such as we find in multiple sclerosis. At times there is a loss of perception of one or another color. It has been asserted that as blindness comes on ataxia diminishes, but the author's experience proves that this is, to say the least, not universally true. None of the special senses are so often affected as that of sight.

Deafness. Vertigo is a common symptom, and it is sometimes associated with hardness of hearing, which is at first unilateral, and only to the high notes, but finally is on both sides, and to notes of all sorts of pitch. It is seen in about 1%, and in the absence of an hereditary tendency, its presence should suggest the possibility of tabes.

Atrophies and Arthropathies. Whether or not there are definite trophic centers in the cord, atrophies always follow a general deterioration of the peripheral sensory apparatus. Gowers says that he has never seen a case of atrophy without a preceding cutaneous anesthesia. These atrophies affect not only the superficial, but also the deeper tissues, just as do the changes

of sensation. Superficially the skin may show glossy skin, eruptions like herpes, pemphigus, and ichthyosis, etc., and there may be subcutaneous ecchymoses, or widespread effusions under the skin. A case is reported in a male, forty-eight years of age, who had a sudden attack of edema of the face, especially of the upper lip, and upper lids. A still deeper condition is that of perforating ulcer, which condition is almost confined to tabes. This occurs mostly in the soles of the feet, but may occasionally affect the hands instead. It may occur in the mouth: first the teeth loosen, then the alveolar margin of the jaw is absorbed, and if it be in the upper jaw, there may be perforation into the nasal cavity. The nails may fall off, and the teeth drop out, all without pain. The deeper effects of the atrophic condition are shown in the fragility of the bones, in which fractures occur so easily that they have been spoken of as spontaneous. Painless fracture should always make us suspect the presence of tabes. The most characteristic of all atrophic conditions is that where the articular surfaces of the joints are disintegrated. This is most common in the knee-joint, and in patients of the female sex. The ankle is the joint affected next in frequency, and the wrist much less often, but the joints of the fingers and toes are exempt. This condition is known as the "Charcot Joint," and its symptoms are the sudden onset of an enormous whitish swelling of the surrounding parts, and absence of pain, disintegration of the joint, and loss of function. Gowers states that in his opinion the "Charcot Joint" is really a rheumatism, which has been neglected, and if, from the first, such cases were treated by rest and local measures, the disintegration would not be a necessity.

Heart Lesions. Nearly all cases of advanced tabes show valvular lesions, especially prone to affect the aortic orifice, but due probably to the initial syphilis, rather than to the present tabes.

Mental Symptoms. These may occasionally be present, not as a distinct psychosis as a rule, but only as a condition of relative apathy. The mentality of the tabetic is usually so clear that he can transact business with propriety, and often with success. There are cases where the cerebral endarteritis, which is a heritage from the original syphilis, produces definite mental changes. These are not always permanent. The case of a woman is quoted who was the victim of expansive delusions, having fainting fits, but no convulsions. A month later she revealed all the classical signs of tabes, and three and a half months later she regained all her mental acuteness.

Rare Symptoms. Lingual hemiatrophy, abductor palsy of the larynx giving rise to difficulty of respiration, rupture of the tendo-Achillis, and muscular atrophies in almost any part of the body. The disease is occasionally complicated by exophthalmic goitre, paralysis agitans, neurasthenia, hysteria, or diabetes.

PROGNOSIS. Cure is in the highest degree improbable, and has been said by many to be impossible, but the disease in itself does not directly tend to death, though their anemic condition, and imperfection of function, continually expose such persons to danger. Cases may generally be expected to reach a condition of permanent invalidism in about ten years, but the grade of invalidism is a very variable condition. The halts between the definite stages, or between the evolution of definite symptoms, may be years in length, and during these remissions re-education of the muscles may make the patient relatively better than he had been before the treatment was inaugurated. There is a small group of cases which may live from ten to thirty years without any manifestation of the disease, except continued attacks of pain. Others show a progressiveness, with changes in the optic nerve, but with little ataxia, or other tabetic symptoms. There is another group where the muscles seem too long and too lax, and allied or combined with it are arthropathies. Then there is a small group where there is an involvement of the motor system, with atrophies of the lower limbs, and with weakened muscles. In the vast majority of cases, ataxia is the prominent symptom, and re-education may neutralize many of the effects of the disease. Those cases with a marked degree of muscular insensibility, and those with a very definite hypotonia, have a bad prognosis. Some cases have been reported cured, and the cure and previous condition have been vouched for by the most competent observers.

DIAGNOSIS. The diagnosis rests upon the presence of Loss of Knee-jerk, Static Ataxia, Argyll-Robertson Pupil, Lancinating Pains, Girdle-sensations, Crises, Changes of Sensation, and Hypotonia. Not all these are present in even the majority of cases, so that we may say that if a case presents lost knee-jerk, and static ataxia it is probably tabes, and that some of the symptoms detailed in the symptomatology will be found to be associated with these, and make the diagnosis certain.

DIFFERENTIAL DIAGNOSIS. Neurasthenia simulates tabes very closely at times, and may be associated with it. A diagnostic difference is the variable character of the symptoms in neurasthenia, and their permanence in tabes. Knee-jerk may apparently be absent in neurasthenia, but care will elicit it, and it may be found at another examination to be exaggerated. An apparent Romberg may be present, but its cause is weakness, and not ataxia, and it will not be evident after rest. Fatigue is also very easily produced in neurasthenia. Apparent crises of the bladder are seen in neurasthenia, but they are due to the irritation of the bladder from the abundant salts from tissue waste, or from neuralgia. Argyll-Robertson pupil, gastric crises, and analgesia are never found, but girdle-pains are seen at times.

Hysteria has many points of similarity, and here the order and

knee-jerk will apparently be lost. It is to be elicited if the examination is a careful one, although it may be greatly reduced. There are hyperesthesias of the trunk, and pain in the back, but these are distinctly worse in the night, which is a syphilitic, and not a tabetic characteristic, and the Argyll-Robertson pupil, and bladder and rectal symptoms are wanting. There is a diagnostic difference in the fact that syphilitic symptoms are rapid in onset, they differ widely in distribution from day to day, or on the opposite sides of the body, and they may be totally absent for a time, and then return again. There are actual palsies also, and these indicate an implication of the lateral columns, or motor nuclei: a paralysis of the iris on one side, or its contraction, indicating involvement of the nuclei of the sympathetic (probably). In all such cases the case is not pure tabes; it is some other disease, or some complicating condition, since oculo-motor palsy of a transient type is the only paralysis commonly attending tabes.

Multiple Sclerosis. In typical form it is not similar, since, while the gait is ataxic, the knee-jerk is exaggerated, pupils are normal and prompt, nystagmus is marked here, and this last symptom is never found in tabes. Lightning-pains are scarcely ever seen, since there are generally no sensory symptoms. Speech is staccato, and there is intention tremor. Confusion may arise when the lesion is in the lumbar cord, since the ataxia now will be combined with loss of knee-jerk; yet even here care will make differences apparent, for the upper extremities will have an exaggeration of the deep reflexes, and the characteristic nystagmus, intention tremor, or staccato speech, one, if not all, will be present.

General Paresis. This disease is at times so similar, that it has often been considered an upward extension of a tabes. The etiology is the same, except that, if the history is searched, it will be found that the brain-worker, as a rule, becomes the parietic, while the mechanical worker has tabes as the reward of his syphilis. Knee-jerk may be lost in both, and the pupillary changes are the same, and both may be ataxic, but the parietic often has an exaggeration of the knee-jerk at first, while the tabetic never has anything but some grade of loss; pains are absent from paresis, usual in tabes and if in paresis at all, they are spinal. The mental state of paresis is most distinctive, since it is an exaltation, or a deep melancholy with a terminal dementia, while the tabetic is only moderately apathetic, or reduced in mental acuteness. Paresis has a marked facial and bodily tremor, and slurring speech; both are wanting in tabes.

Paraplegias. At the first glance, the paraplegias are similar to tabes, but the differences are definite. In the ataxic form with a pronounced degeneration of the posterior columns, the difference is, that while the patient is actually an ataxic, he has no pains, and the deep reflexes are exaggerated. In the spastic form, not

sequence of symptoms is often the best guide. There is never loss of knee-jerk, nor is there an Argyll-Robertson pupil, nor optic atrophy.

Chronic Myelitis. This is a true paralysis, and not an incoördination. Atrophy is immediate and pronounced, and is not of a mild type and general character, but is localized and extreme in those parts innervated by definite segments of the cord. There is pain in the back in a definite area, and the knee-jerk is exaggerated, unless the myelitis is in the segments making up the reflex arc, when it is lost. There is no disturbance of the special senses, but the sphincters are incontinent.

Hereditary Ataxia develops in the period from puberty to the twentieth year, while *tabes* is from the fortieth to the fiftieth years, except in the rare cases where hereditary syphilis is the cause. Friedreich's type has a loss of knee-jerk, (in Marie's it is increased), and the patient has a great deal of static ataxia, but no Argyll-Robertson pupil, or other eye-symptom, except almost certain nystagmus (oscillation of the eyeball in certain positions), which is the one eye-symptom that the tabetic *never* suffers from. He has an apathetic mental state, somewhat like that of *tabes*, but he never has the lightning-pains, or bladder symptoms of *tabes*, but he does have contractures, talipes, and claw-hand, which we do *not* find in *tabes*.

Multiple Neuritis. This may complicate, and it then makes the case contradictory in its symptoms. There are also cases with much ataxia, and little palsy, which are also difficult. Pure cases however strike us at once as a palsy, and not an incoördination. A history of some toxemia, or of some recent infection, can generally be obtained, and the onset is rapid, while that of *tabes* is conspicuously slow. The bladder and rectum are rarely, but certainly not permanently affected in multiple neuritis, but such disturbances are early and usual symptoms in *tabes*. Pains of a lightning character, crises, and cuirass-sensations are absent, and if there is anesthesia, it is of a glove and stocking-type, avoiding the trunk and face, and not widely distributed in strips and patches as in *tabes*. If the Argyll-Robertson pupil is present, it stamps the case as something other than multiple neuritis, but if the pupil will react neither to light nor to accommodation, the diagnosis is doubtful, until the lapse of time shows that it is not multiple neuritis. There may be root-pains in multiple neuritis, and there may be ocular disturbances. Early muscular wasting, and the reaction of degeneration are not tabetic, nor are the nerves swollen and tender to touch in that disease.

Syphilis of the Spine, Syphilitic Pseudo-Tabes, and Subacute Syphilitic Meningitis. These are conditions which are more nearly allied to *tabes* than any other, since the patient may actually have lightning-pains, paresthesias, and ataxia, and the

knee-jerk will apparently be lost. It is to be elicited if the examination is a careful one, although it may be greatly reduced. There are hyperesthesias of the trunk, and pain in the back, but these are distinctly worse in the night, which is a syphilitic, and not a tabetic characteristic, and the Argyll-Robertson pupil, and bladder and rectal symptoms are wanting. There is a diagnostic difference in the fact that syphilitic symptoms are rapid in onset, they differ widely in distribution from day to day, or on the opposite sides of the body, and they may be totally absent for a time, and then return again. There are actual palsies also, and these indicate an implication of the lateral columns, or motor nuclei: a paralysis of the iris on one side, or its contraction, indicating involvement of the nuclei of the sympathetic (probably). In all such cases the case is not pure tabes; it is some other disease, or some complicating condition, since oculo-motor palsy of a transient type is the only paralysis commonly attending tabes.

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only are the knee-jerks exaggerated, but there is ankle clonus also, and there is no ataxia, but in its place there is much weakness. In cases arising from a complete division of the cord, there is absolute paralysis in all parts of the body below the lesion, while such a loss of motion is not found in tabes, up to the last moment. There are changes in sensation in definite areas in tabes, while they are absent in the spastic, and often in the ataxic forms of paraplegia.

Cerebellar Tumor. Both this and tabes have ataxia, and occasionally the knee-jerk is absent in both, but as a rule the latter is exaggerated in cerebellar tumor. There is a tendency to fall in both, but it is definitely to one side in most cases of cerebellar tumor, and there may be jactitation of the feet. The ataxia of tabes pertains to the erect position, while it is just as evident when the tumor patient is lying down. He is much more liable to choked disc, his head is retracted, and he has occipital pains. The tabetic alone has the Argyll-Robertson pupil, lightning pains, sensory and bladder disturbances.

Syringomyelia. The lower limbs are generally paraplegic, but they may be ataxic, and the disease is generally seated in the upper part of the trunk, and in the upper limbs, which are usually spastic. Characteristically, with otherwise normal sensibility, the loss of the sense of temperature is the primary symptom. Muscular atrophy, painless whitlow, necrosis of the phalanges, and lateral scoliosis are present in syringomyelia, and are absent from the symptomatology of tabes.

TREATMENT. The subject of treatment must be approached with a thorough knowledge of the pathological condition, which we have gone over with some particularity, and also with the appreciation of the value of psychical influences upon a patient. It may be broadly stated that, so far as we know, a nerve-cell once destroyed is never reconstituted. Remembering that this is a disease finally degenerating the cells of the column of Clarke, and of the posterior root-ganglia, we can see that when that stage has been reached, we have to deal with a process impossible of cure in the ordinary sense of the word. That is: we cannot hope to heal the scars in degenerated tissues, and we know that what has been destroyed will never be regenerated. When once the nucleus has passed out of the cell, that cell is dead, and it cannot be regenerated. We must remember, however, that the cells energizing a given nerve are myriad in number, and that while all may be affected, all may not be destroyed. A part may be destroyed, and a part may be only functionally disordered by the toxic products of the general process. Munk has stated that in some autopsies there have seemed to be signs of a partial regeneration of nerve-fibres in degenerated areas, which may indicate that under favorable conditions the enfeebled, but not destroyed cells may

have taken on a new vigor, which has been reflected in renewal of the structures of the nerve-fibres. This regenerative process has never been seen to exist in more than a slight degree, and clinically no facts have been advanced by competent observers confirmatory of it, except that some cases do recover. • The wonderful cases of cures by this lymph, that shoe, or that regimen, or remedy, have always rested under the imputation of faulty diagnosis, or of being a too optimistic interpretation of one of the well-known halts, and apparent recessions, of the disease. There is no doubt however that the mere idea that treatment is being carried on has a powerful effect in retarding the progress of the disease.

Rest in bed is of the very first importance ("Before all else," says Ormerod), for a period of six weeks or so at a time. (This is the exact contrary of the treatment in multiple sclerosis, and the spastic forms of paraplegia, where it may result in making an ambulatory case a bedridden one). There should be immunity from strains and over-exertion, particularly those contributing to leg-weariness. General treatment should be directed (1st), to relieving any active syphilitic process. This is generally one of the conditions that has passed; yet, mixed up with the actual tabetic symptoms, we may find others, cerebral, neuritic, myelitic, and osseous, which are syphilitic, and may be relieved by proper treatment. Erb always advises this "to clear the air." It must be kept in mind however that the administration of mercury is not always devoid of harm. (2d) Improve the general health as much as possible. Rest is one of the measures, and plenty of sleep; a careful selection of food, and the cutting off of stimulants and tobacco. Hydrotherapy has been advised in the form of cold douches, steam douches, hot needle-baths, and affusions, and sponging the spine with hot and cold water. It is now thought to be of doubtful utility, and Oppenheim says that while it does relieve the pains; it accelerates the progress of the disease as a whole. See that the bowels and skin are kept in good condition. A change of air occasionally, and plenty of fresh air while at home should be insisted upon. A mild climate is to be given to those who can afford that luxury; some exercise, but mild in character, and without monotony. (3d) Improve the nutrition of the cord. This is accomplished by measures which increase its blood supply, and elongation of the spine is the most efficient method. An early method was suspension of the patient for four or five minutes in a Sayre's apparatus, but that was dangerous, and it has been superseded by the method of Tourette and Chipault. They proved that the cord could be stretched by a forward bending of the body. The procedure is carried out by seating the patient on the floor, or a hard table, with the lower limbs extended, and held together in front of the body. The head is then forcibly depressed toward the knees, which are not allowed

to bend. The full degree of extension should not be produced until two or three partial extensions have been performed on successive days. When the patient has become able to permit the head to touch the knees, the position should not be maintained for more than two minutes at a treatment. (4th) Maintain the muscular control so far as is possible. In the pre-ataxic stage the patient should practice the movements in which he shows deficiency, while if in an advanced stage, he should be given a full course of the movements prescribed by Fraenkel. Fraenkel (Berliner Klinische Wochenschrift, June 5th, 1905) says that his teaching in regard to exercises has been a failure, because overdone. Restrict the patient to two sessions a day of not more than 5 to 15 minutes each. The patient has lost his perception of fatigue, and so his feelings are no guide. The trunk muscles are the originators of the acts of locomotion, and an exercise of them and a re-education of them is valuable, and has reduced the number of cases of atony of the abdominal muscles, and of the intestines, and also of cystitis. Weakness of the sphincters is best treated by the method of Brandt. While lying on the floor, or a hard bed, the patient should abduct, and adduct, the knees successively against pressure, and at the same time should vigorously draw up the pelvic floor. This procedure should be repeated about twenty times daily. Massage of the perineum is also of value.

Electrical Treatment. The best treatment where galvanism is used is to give an ascending current along the spine, placing the negative electrode on the back of the neck, or just under the ears, and the positive, stable in the lumbar region, continuing the current from ten to fifteen minutes with a strength of from 10 to 25 milliamperes, painting the spine with iodine before each application. Then place the anode on the abdomen, and the cathode, stable on one segment after another, taking about twenty minutes for the application. This will relieve very greatly the lightning-pains. Static electricity is very effective, if severe sparks are drawn from the spine and roots of the spinal nerves for at least three to five minutes; then milder sparks from various parts of the body following down the trunks of the nerves. Draw sparks from the bottoms of the feet for from two to three minutes, which reflexly affects the spine, improves the conductivity of the nerves, and has a great effect in temporarily relieving the ataxia. In the early stage galvanism helps, but static irritates; in the second stage where there is slight ataxia, galvanism is still the better. Where locomotion is very difficult, and the case is complicated by interference with the functions of various other organs, static is superior to galvanism. Many see a profound stimulation from the high-frequency currents in use to-day. Nutrition of the spine is also increased by flagellation of the back with a scourge, made by

attaching a broad, stout piece of leather to a short handle. This may also be used to advantage on the soles of the feet, and the palms of the hands, and also on any anesthetic spots.

Counter-irritants have been used for a long time, but whether they act by suggestion, or really cause an increase of circulation to the part, I am unable to say. In some way or other they often seem to do good. The best method is the application of the cautery of Pacquelin. Bring it to a white heat, and make dots along the spine over the lateral processes. If it is at white heat, the application is quite devoid of pain. If nerves are affected, it may be applied along their course. In the use of the cautery it is best to repeat it at such intervals as not to make the skin an open sore, and give 6 to 10 applications, and then wait a month or two before making another series of applications. Richardson recommends a cathode $1\frac{1}{2}$ inches wide by 5 inches long, bridging the spinous processes, but not in contact with them. It is the active pole, and should be placed over the cervical spine. The anode is the other electrode, at least 3 to 5 inches long, and placed upon the sacral region. A current of 15 to 30 milliamperes is passed for three minutes, then turned off, and the active pole moved its length downward, and the same current used for the same length of time. The process should be repeated till every part of the spine has been covered once, and in bad cases twice, by the treatment. The strongest current should be used that can be borne, and that is usually about 30 milliamperes. He regards this as curative in the peripheral, or benign forms of tabes.

Carbonic Acid Baths. Jean Heitz thinks that great benefit has come from carbonic acid baths to the number of 20 to 25, as they give them at Royat. It was shown in general condition, improvement in superficial and deep sensation, plantar sensation particularly; analgesia disappeared along the course of several nerve-trunks; stereognostic sense and that of position became more accurate, and ataxy less marked. He thinks that it acts by tonic effect on the myelin sheaths, and that it will keep a patient at his avocation longer than the Fraenkel movements. Schwab and Allison say that the tabetic foot is a pronated foot. The effect of this pronation leads to muscular strain on the ankle, knee, hip, and spine. This, with the hypotonia, tends to break down the long arch, and makes a faulty apparatus with which to walk. If then we remedy this defect with a proper shoe, and make use of the Fraenkel method of re-education, we can give a great deal of aid to the tabetic.

THERAPEUTICS. The indications and clinical reports for the use of remedies are in many cases to be viewed with a great deal of suspicion, since a careful analysis will develop the fact that many of the so-called ataxias are due to other conditions than lack of sensory innervation, and many of the paralyzes are dis-

tinely spastic, while, on the other hand, pains give every indication of having been caused by rheumatism, instead of having been caused by irritation of the posterior nerve roots. Granting all this, and allowing that many indications are *sub judice*, there is no doubt that by the careful selection of remedies according to the rule of similars, many cases upon apparently slight indications have been greatly benefitted, and, while perhaps not cured, have been insured long periods of remission from painful or disabling symptoms. Physiological medicine, except in the one department of the relieving of pains, has not been any more favorable, and in my own experience seems to have been far less beneficial to the patient. Another point should be mentioned, and that is that remedies having a toxicological basis like *Lathyrus*, *Secale*, and *Aesculus*, have not given the amount of relief which might hopefully be expected.

Aesculus hip. This drug has produced in sheep an absolute tabes dorsalis verified post-mortem. It gives rise to a general paralytic feeling, with great weakness and lightning-like pains in the lower extremities. A characteristic symptom is a spasmodic contraction of the rectum, and if present in the disease should be an indication for the use of this drug.

Alumina. Apparently acts especially upon the muscle-spindles, and therefore we find anesthesia and paresthesia of the soles of the feet, and also quite generally distributed over the body, and even in the face. The use of limbs seems to increase the subjective sensations of numbness; constipation is marked. The mental change is one of apathy.

Angustura vera. Is one of the doubtful remedies, since its symptoms are those of a spastic instead of an ataxic paraplegia, but O'Connor reports the relief of lightning-pains in two cases.

Argentum nit. The same criticism of this remedy has been made as of the preceding. Its ataxia seems far more dependent upon some form of multiple neuritis, or ataxic paraplegia, than upon tabes, but Allen considers that some cases have been cured by it.

Belladonna. Indicated for lightning pains which have the characteristic of suddenness of onset and disappearance.

Cadmium met. The toxicological reports of this remedy show that it produces degeneration of nervous tissue, and its indication is cutting and lancinating pains like Zinc.

Carboneum sulphuratum—Carbon bisulphide. The use of this remedy is based upon its toxicology, reports of which are rather extensive, since it is a universal solvent for rubber. There are marked formication and numbness in the hands, with pains in the lower limbs, with cramps, trembling, and ataxia, the causation of which is doubtful. There are some clinical reports of its efficacy in the treatment of lightning-pains.

Causticum. Has been suggested, but its particular action is upon nerve-cells, which is not the point of attack in tabes, and the paralysis which it causes has not the distinctive marks of tabes. There is a condition of tension in the muscles instead of relaxation, as we should expect from the pathology, but there are paresthesias of the extremities, and it may prove to be useful in some cases.

Colchicum. Certain cases of tabes are marked by pains only, and with very few symptoms of motor disability. Cases are known to be precipitated, at least apparently caused, by exposure to cold and wet, and in such cases, Colchicum should be useful.

Cuprum ars. and met. Are marked by muscular cramps and lightning-pains, and have often been very efficient in relieving these symptoms in the early part of a tabetic seizure. There is a great tremulousness of the whole body on attempts at walking.

Duboisia. Has weakness in the limbs, and heaviness in them, with pain in the lumbar region, and a great deal of ataxia. If diplopia is found in such cases this should be a good remedy.

Fluoric ac. For types with great sexual excitement, and with all symptoms excited by cold and wet. There is much paresthesia and subjective numbness in all the limbs.

Gelsemium. Has been considered to be a remedy for functional rather than organic conditions, and the tremulousness, neuralgic pain, and cramps in the muscles are irritative symptoms. If hyperesthesia of the surface is a marked symptom, which is unusual, Gelsemium will avert the anesthesia which will be the result.

Graphites. Has no particular grounds for use except as an absorbent, but like some other remedies has been credited with cures.

Kali brom. It has great ataxia, and loss of perception of touch, pain, and temperature. It is considered to also produce an increase of sexual appetite, priapism, but without ejaculation.

Lachesia. Has been credited with cures, but the symptomatology shows a decidedly spastic form of paraplegia.

Nitric ac. Is marked by burning superficial pains, *plaques douloureux*. Some cases of cures from its use have been reported.

Nux moschata. Has been held in great repute. Lippe marks it as his first remedy. The limbs are painful, with an aggravation in cold and damp weather, and there is a numbness in the limbs.

Phosphorus. Is Lippe's second remedy, and should be thought of in cases where, with great sexual excitement, there are burning and formication along the spine. There may be flashes of light before the eyes, followed by optic atrophy. A sexual excitement in the beginning, or at some period, is a *sine qua non* for its use.

Picric acid. Has anesthesia and subjective numbness of the extremities, but its main indication is a high degree of priapism. All these cases presenting this symptom would indicate an involve-

ment of the cervical portion of the cord. This is a very unusual localization, but if present one of these remedies should suggest itself.

Plumbum. This remedy has been highly recommended, and is said to be applicable to advanced cases where there is great sclerosis. There are numbness in the feet, and convulsive movements in the limbs, and while therefore it seems to affect the whole cord, it may be useful in cases where constipation is a marked symptom, and paresthesias of the extremities.

Secale. There are absence of knee-jerk, fulgurating pains and, ataxia. There is no doubt but that eating of spurred rye has produced degeneration of the posterior columns of the cord, and while the results of its use have not been satisfactory, either when used in material doses, by Physiological therapists, or in potencies by our own practitioners, it should still be kept in mind as a possible resort.

Silicea. Cures have been claimed for this drug, but no distinct symptoms can be stated, and it must be prescribed upon general constitutional conditions.

Strontium carb. Has some indications which point to girdle sensations somewhat suggesting the tabetic cuirass. There is a paralytic weakness in the limbs, but it is to be noticed that there are symptoms of tension, which might be from root irritation, which would be the source of the preceding symptom also. There is a subjective numbness on the limbs, and peculiarly also on the face. All the symptoms are very much aggravated by cold, and ameliorated by warmth and wrapping. O'Connor mentions that one case was very much relieved of lightning-pains by it in the 30th; the indication being that they were only relieved when the parts were in hot water.

Syphillinum. This is suggested from the fact that the disease is probably the result of the action of a late toxin of syphilis. Favorable reports have been given of the action of this remedy in the higher potencies. The author's experience has been that marked remissions have certainly taken place under its use.

The literature of Physiological Medicine is very voluminous, and fully as unsatisfactory as our own, but the treatment of the pains certainly demands at times the use of all sorts and kinds of expedients. Hypodermatic injections of Morphine will sometimes be the only refuge, and some have said that in this condition there is no danger of forming a habit, but this possibility must be kept in mind. The most certain results are to be obtained by the use of the coal tar-products. In order of efficiency they are Anti-febrine, Antipyrine, and Phenacetine. Antipyrine may be given in doses of 10 grs. hourly during the period of most acute pain, but as the pain subsides it should be given only three or four times a day. Bartlett says that 3 grs. given hypodermatically will

relieve the spasm of pain in three minutes, but never give more than 5 grs. as an initial dose, as it is extremely irritating to the tissues. Sometimes benefit will be received by giving a thoroughly triturated powder of Acetanilid one week, Antipyrine the next week, and Phenacetine the following week. Ten grains of Phenacetine with 1-60th gr. of Strychnia sulph. will sometimes give relief, when any one of the analgesics alone will fail. A teaspoonful of baking soda internally will sometimes stop the pains. Cannabis ind. 1-2 to 1-4 gr. every three or four hours so long as the pain persists. For the superficial pains put chloroform on lint, and cover it with oiled silk, or inject Cocaine very superficially, for if it is injected into the deep muscular masses it will do no good.

Bladder and Rectal Pains. Young says, give

Suppositories. Pulv. Opii. Gr. 1.
Ext. Belladonna. Gr. $\frac{1}{2}$.
Iodoform. Gr. 3.
Ol. Theobromae. Grs. 30.
Misce et ft. Suppository No. 1.

Santonin. Bricage calls attention to this drug. M. Collet gives grains 5 to 7 in 3 doses a day. For avoidance of the toxic effect do not go beyond this, or give $2\frac{1}{2}$ to 5 grs. continued for weeks. The lightning-pains are controlled by this, and the relief continues after the cessation of the drug. On account of its feeble toxicity, it may be continued for two months. It will also relieve the laryngeal crises, but it is powerless against neuralgia or sciatica.

Sodium nitrite. Doctor Raymond says that mercurial injections will not always cure lightning-pains. Do not continue them longer than six weeks. If no benefit results, change to hypodermatic administration of 1 c.c. of 1% solution of Sodium nitrite; give once a day for ten days, then suspend it for ten days; resume for ten days, suspend it again for ten days, resumed with a 2% solution for ten days; rest for ten days, resumed with a 3% solution. In all, the treatment to be continued until forty or fifty injections have been given, and not before this can much relief be expected, although in the case of a young woman, for three years a sufferer with lightning-pains in the legs, unrelieved by mercury and other anti-syphilitic treatment, a marked improvement was noticed by the 24th injection.

SPASTIC PARAPLEGIA.

PRIMARY LATERAL SCLEROSIS.

DEFINITION. This is a chronic, progressive degeneration of the spinal cord, which occurs in adults, with a weakness of the legs which is accompanied by a stiffness of those members. Its occurrence, as a separate entity, has been doubted, but Erb, who first isolated it, has recently reasserted his original proposition.

THE SYMPTOMS OF A TYPICAL CASE. An adult will gradually develop a weakness in the lower limbs, with an exaggeration of the deep reflexes, which condition will finally render the person helpless, on account of weakness and rigidity. There will be ankle clonus, and Babinski's reflex, when the condition has become marked, but from first to last there will be no sensory changes.

DIFFERENTIATED from Syphilitic Meningo-myelitis. Pseudo-paraplegic Rigidity. Multiple Sclerosis, Chronic Myelitis. Amyotrophic Lateral Sclerosis, Hysteria.

AGE. It occurs in adults from the thirtieth to the fortieth years. It may occur in children in the pure form, but in them it is generally cerebral, and from prenatal causes. There is a family type occurring in young people from the twentieth to the thirtieth years, but usually not in a pure form, but in combination with a simultaneous degeneration of the posterior columns, and the brain.

ETIOLOGY. In the pure form under discussion it seems to result solely from a selective action of an infection, or from traumatism. Cases have been isolated in India which were caused by poisoning by *Lathyrus sativus*, or Chick-pea.

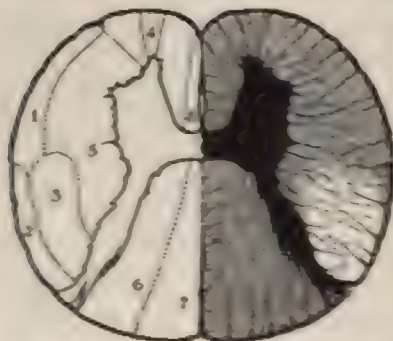


FIGURE 30.—SPASTIC PARAPLEGIA.

Observe that the degeneration shown in white in the right half is in the crossed Pyramidal Tract (5) seen on the left half.

PATHOLOGY. There is a primary degeneration of the lateral columns of the cord, with slight implication of the uncrossed pyramidal tracts. The same symptoms, so far as the lateral columns are concerned, may arise from compression of the tracts, but other tracts could hardly be expected to escape injury at the same time. The infantile, adolescent, and familial cases, all arise from a primary de-

generation of the brain, which is followed by a degeneration of these tracts, or from a deficient development of them (agenesis).

SYMPTOMATOLOGY. The first symptom to appear is a weakness of the lower limbs, but the patient is apt to explain that to his own satisfaction as being due to some acute condition. The rigidity of the limbs, which appears later, by its combination with the weakness, is apt to so embarrass his locomotion that he will seek an explanation from the physician. If then questioned, it will generally be found that he has been experiencing a sensation of weakness for a considerable time. Inspection at this time will reveal the fact that the toes of his shoes have been worn off in an unusual manner, his gait will be clumsy, and he will recall that he has been stumbling unaccountably of late. The deep reflexes will be found to be exaggerated, the knee-jerk is over-active, there is ankle-clonus, and a Babinski can be elicited. This consists in an over-extension of the great-toe certainly, and probably of all the toes, when the sole of the foot is stroked from the outside inward ending at the base of the great-toe. When the reflex is sluggish, the use of an instrument, with a somewhat sharpened extremity, may cause the reflex to appear more decidedly. The over-extension of the great-toe is all that is absolutely essential to determine the presence of the reflex. There is no wasting of the muscles, nor is the reaction of degeneration present. There is no loss of sensibility of the tissues, and pain is absent, except that there may be a little in the muscles if the process is a rapid one. The limbs are the seat of painful spasm, particularly at night. The functions of the bladder and rectum are normally performed.

COURSE. It is a disease of slow progress, and it may be twenty years before the whole symptom-complex is evident. It may come to a standstill at any stage, or, after a long remission, again progress. For a long time, a spastic paralysis of the lower limbs may be the only departure from health, but in the full-developed disease, the arms finally become involved in a similar manner, and at times the face shares in the paralysis.

PROGNOSIS. Cures of definitely outlined cases are unknown, but the progress is so slow, remissions are so common, and the symptom of spastic paraplegia is so often only the preliminary stage of some other disease, that a definitely bad prognosis is uncalled for. If, after a time, we are convinced that it is lateral sclerosis, we can assure our patient of the slight danger to life, and a possibility of only a relative amount of disability. In severe and widespread cases, we can only hold out a hope of the continuance of the present condition.

DIAGNOSIS. This rests upon Weakness of a moderate degree; Spasticity (especially of the lower limbs); Exaggerated reflexes (deep); Absence of sensory symptoms, or of disturbance of the sphincters.

DIFFERENTIAL DIAGNOSIS. Syphilitic Meningo-myelitis. This gives rise to a spastic paraplegia, but it comes on abruptly,

and the degree of paralysis rapidly increases. It does not confine itself to the lateral columns, but gives rise to pains and other changes in sensation, and disturbs the functions of the bladder and the rectum. It has a grave prognosis.

Pseudo-paraplegic Rigidity. This is a condition found in children who have suffered from rickets, laryngismus stridulus, and the so-called hydro-cephaloid state. It begins in the hands instead of the feet, and is often confined to the hands and arms. It gives rise to spasms, instead of a relative rigidity, and attempts at reducing it by extension give rise to pain. It is also intermittent, and of transient duration.

Multiple Sclerosis. This is the disease for which spastic paraplegia is most often an initial stage. It may begin, and remain, for a time, a simple spastic paraplegia. Some sensory symptoms are possible, however, but more especially the early advent of some cerebral symptoms, vertigo, ocular palsy or nystagmus, will differentiate the two conditions. Mental changes are common in multiple sclerosis, and are unknown in spastic paraplegia. If typical, multiple sclerosis is not at all confusing, since nystagmus, scanning-speech, intention tremor, and ataxia are not found in spastic paraplegia.

Chronic Myelitis. This produces a spastic form of paraplegia, and therefore the initial periods of both may be similar, and this points out the necessity for conservatism in the diagnosis of spastic paraplegia. Like at first, they very speedily diverge, and myelitis alone has root-pains, pains and hyperesthesia of the spinal column, and of areas of skin on the abdomen, and often on the legs; girdle-sensations. (Pain in the spine is often an initial symptom in myelitis, while it is never present in spastic paraplegia.) The bladder and rectum are disturbed, there are bed-sores, and deformities from contractures.

Amyotrophic Lateral Sclerosis. This is a spastic form of paraplegia, but while the lower limbs are spastic and paraplegic, the upper limbs are apt to display atrophies at an early stage of the disease, which are finally very marked in the upper or lower extremities, or both.

Hysteria. This may exhibit a spastic form of paralysis, but sensory symptoms are always present, and generally of a distinctive sort. The rigidity of hysteria is peculiar, in that the efforts to reduce it will be antagonized by a fixation of the extensors, as well as the flexors. The leg is steadied, as it were, in a determined position. When it succumbs to irresistible pressure, it gradually gives way, while the rigidity of spastic paraplegia is "lead-pipe," i.e., after a short period of resistance it suddenly doubles up, and is perfectly flexible. Chloroform narcosis will also render the hysterical rigidity perfectly flexible.

TREATMENT. The main reliance should be upon hygiene, nutri-

tion, and stimulation of the nervous system. The patient should therefore lead a regular life, and, while using all means to promote muscular nutrition, should avoid over-exertion. The diet should be rich in fats, and cod-liver oil, and the petroleum emulsions, with or without the aid of hypophosphites, should be a part of his diet. There is a very great divergence of opinion as to the amount of rest that should be taken. Some advise a month's rest in bed as preliminary to other treatment, and assert that it has started some cases toward remissions, who had, under other methods, been steadily becoming more and more incapable. On the other hand it is the observation, in places where large numbers of such cases are treated, that a month's entire rest may make a bedridden invalid of a person who was previously able to go about enough to earn his own living. The weight of opinion is in favor of moderate exertion, and the practice of the same exercises as are recommended in tabes. The aim of these exercises is the re-education of the spastic limbs, and has strong testimony in its favor. Stimulation by faradism and strychnine is condemned by all but a few. Its advocates acknowledge its theoretical danger, but urge occasional brilliant results. The relief of the spasticity is sometimes accomplished by warm baths, but still more efficiently by the Turkish-bath, or sweating, or warm or hot douches to the spine and limbs. Massage should be given in an upward direction, while the limbs are held to such a degree of extension that the muscles are tense. This is also good for the relief of the spasms of the muscles at night, which are so painful. For this is also used the spinal ice-bag. The warm bath should be from 90 to 95 degrees. One author, copied by many others, has said that galvanism is useless, and faradism is positively harmful, but King says, cures reported of the early stages were too early to allow the diagnosis to have been established; it is, however, a palliative for the spasticity of the muscles. The strong static spark over the spine and nerve-roots is beneficial, but not over the muscles, except when paresis has become marked. It will then relieve by invigorating them, as will faradism. This is especially true of the quadriceps extensor. Galvanism may be used over the spine for eight minutes, with a descending current of twenty to twenty-five milliamperes, electrodes three inches in diameter. Put the anode now on the abdomen, and galvanize each segment with the stable cathode for three minutes, following with two or three interruptions on each segment. To relieve spasmodic rigidity in muscles, use the positive with a long, narrow electrode over the muscle, retaining the cathode on the spine. Raise the current gradually to ten milliamperes, and allow it to remain there for two or three minutes, and then as gradually reduce it. Go over every set of muscles in this way. Interruptions over motor points should not be made except very late in

the disease. Steavenson of London places patients in a bath to which the cathode is attached, the anode being on the spine between the shoulders. Ten to fifteen milliamperes are given, for from fifteen to twenty minutes. Some, he says, are much invigorated, and the spasms reduced, while many are not helped.

THERAPEUTICS. The outlook from remedies is well-nigh hopeless, but, since some cases recover whose symptomatology is distinctly that of spastic paraplegia, it is well to do all that is in our power to possibly abort a disease, which is incurable in the chronic stage. Since this is not a cellular disease (we know that cells once destroyed are never regenerated), it may be possible that patches of sclerosis may be absorbed. An incipient case was totally relieved by hourly doses of Manganese binoxide, 3x. Strychnia phos. 3x. is sometimes of benefit, and if such a potency aggravates the symptoms, a higher one may be useful. Mercury is always a possible remedy in sclerosis of nervous tissue. Many other remedies are advised, but their symptomatology is not free from the presence of sensory symptoms, which would seem to render them inapplicable. The distress from the muscular twitchings of the legs, worse generally at night, often baffles our best endeavors. Belladonna is not good, since it has sensory symptoms, numbness and shooting pains. Lathyrus produces a spastic form of paralysis, with exaggeration of the reflexes, without any sensory symptoms, except muscular cramps. It has vasomotor symptoms, shown by blueness and coldness of the extremities. A cure by it in the 3x. is quoted in the case of a hunchback of twenty-eight, but it is to be noted that he had a girdle-sensation, as though a cloth wrung out of cold water were around his waist. The general testimony seems to be that Lathyrus does not cure spastic paraplegia, and the case above quoted, presenting a girdle-sensation, shows that it was a case of meningeal myelitis, probably initially from compression, which was followed by extension to the meninges. Manganum has too many sensory symptoms to be a theoretical remedy for spastic paraplegia. Phosphorus has paralysis, but it is always marked by sensory symptoms. Plumbum has the same. Ustilago maidis also has pains. Causticum has subjective sensory symptoms. Arsenicum has numbness and tingling. Argentum nit. has spastic paralysis in the legs, with weariness and debility, stiffness, cramps in the muscles at night, rigidity, tearing pains in the muscles, and in the nerves. The symptoms seem to be more ataxic than paralytic, and there are pains and numbness. Zinc has heaviness, stiffness in the legs, nervous movements when lying down, and in sleep, but there is marked sticking and burning pain. The stiffness and spasm seem to result from the neuritic condition, more than from the central condition. There is paralysis from exhaustion, and auto-intoxication. There is constant

reference to pains in the symptomatology. *Nux vomica* is advised, but the symptomatology has many references to disturbed sensation, pain, numbness, and formication. *Graphites*, *Hepar*, and *Silicea* should be thought of for their efficiency in promoting absorption.

Of the physiological remedies, a trial of Bromides, and other sedatives, has developed a rather general agreement, that *Anti-pyrin* is the best.

CHAPTER IV

COMBINED SYSTEM DISEASES

ATAXIC PARAPLEGIA.

POSTERO-LATERAL SCLEROSIS.

DEFINITION. This is a rather common form of nervous disease affecting the cord by a sclerosis of the posterior and lateral columns. It gives rise to a paraplegia, in which the symptoms of spasticity and ataxia are combined to a variable degree, some patients being very spastic, while others are equally ataxic.

THE SYMPTOMS OF A TYPICAL CASE. There is a spastic paralysis of the lower limbs, with an increase in the deep reflexes, but with some sensory changes, and later a pronounced ataxia appears, which is followed by a diminution, and perhaps a total loss of the deep reflexes.

DIFFERENTIAL DIAGNOSIS. Tabes, Multiple Sclerosis, Spastic Paraplegia, Myelitis, Family Ataxias, Cerebellar Tumor.

AGE. In general it is a disease of the period of most vigorous life, i.e., from thirty to forty, but has been seen as early as fifteen, and as late as sixty-one.

HEREDITY. This has been asserted to exist in one-tenth of the cases.

ETIOLOGY. It results from a very wide range of causes. Tabes may cause it, and general paresis still more often. It has followed an attack of myelitis, and also lepto-meningitis of the cord, which has set up a marginal form of myelitis. Specific and general intoxications have caused it, like pellagra, and syphilis, although it follows syphilis as seldom, as tabes does frequently. It has followed directly upon muscular over-exertion, and upon exposure, particularly after such exertion. It has arisen without any assignable cause, and therefore there is strong probability that embryonic deficiency, or localized nervous vulnerability lies at the root of all cases.

PATHOLOGY. It is a sclerosis resulting from an endarteritis of the spinal vessels, and varies in vertical extension according to the duration of the disease. The pia has been found adherent to the posterior and lateral aspects of the cord. The sclerosis is usually confined to the distribution of the posterior spinal arteries. The

dorsal region is the first attacked, and the lumbar region may be spared to a great extent. In long-standing cases the region of the anterior arteries is invaded to some extent, and the column of Clarke and the anterior spinal roots have been degenerated, but complete destruction of any definite tract is rare, and the posterior root-zone is very rarely involved (opposite of tabes). The complete picture is a degeneration of the column of Goll throughout its whole extent, column of Burdach above the lumbar enlargement, crossed pyramidal tracts only slightly, direct cerebellar very severely, while the column of Gowers' often escapes entirely.

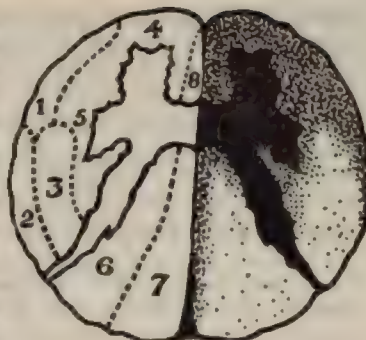


FIGURE 51.—ATAXIC PARAPLEGIA.

The degenerated portions appear as white areas in the right half. They may be identified by reference to the schematic left half. They are the column of Goll (7), of Burdach (6), the Crossed Pyramidal tract (3), and the Direct Cerebellar (2).

SYMPTOMATOLOGY. As has been said, it is a combination of an ataxic and a spastic paraplegia, and not at all uniform in the proportion of either form of disability, but usually it first becomes pronounced as a spastic form of paraplegia, complicated by a very definite ataxia. Since the direct cerebellar tracts are so uniformly and so severely injured, we are sure to find a high degree of ataxia and incoördination, and the well-nigh universal sclerosis of the posterior columns indicates that sensory disturbances will be prominent. We are sure to encounter some degree of anesthesia and paresthesia, but lightning-pains are almost unheard of. Some sacral pain is diagnostically present, and is also apt to be in the legs. The knee-jerks are exaggerated at first, but generally diminish at a later stage. The knee-jerks are preserved, because the removal of inhibition by disease of the lateral columns allows stimulation of anterior horn cells, and production of the reflex, even with impairment in some part of the arc. Ankle-clonus is generally present, but Romberg's sign and an ataxic gait are always present to some degree. The disease usually ascends the cord, and the upper extremities become spastic, and also incoördinate, and there are irritation symptoms of the bulbar nuclei, i.e., twitchings of the face and tongue, and thickening of speech, and some ocular symptoms, viz., reflex accommodation being lost, that to light remaining; optic atrophy (syphilitic cases), and nystagmus on movement. The mind is rarely affected, and, if so, only to a slight degree of memory defect. Owing to the involvement of the lateral columns, there is a degree of muscular weakness proportionate to the involvement of those tracts, and

a tendency in all muscles to cramp-like spasm on use of them. There is no marked atrophic change, and while cystitis may occur, it is from retention owing to the spasticity of the sphincter. Sexual power is often lost early in the disease. The combination of symptoms does not follow such a regular order as is common in system diseases, and while those just enumerated may be present, many may be lacking, some may be added (arising from the primary and exciting disease), but, in order to make the disease come under these captions, there must be some combination of spastic paralysis and ataxia. In a case now under treatment there is a history which may be illuminating. A man probably acquired syphilis at the age of about thirty-two, and since then has had repeated prostrations, which have been attributed to nervous exhaustion from business cares. In 1890 he had a suddenly developed ptosis of the right eye, lasting thirteen weeks; at the same time retention of urine, some vertigo, weakness of the lower limbs, and marked ataxia. At the present time he has both knee-jerks exaggerated, the left more than the right, ankle-clonus on both sides, Babinski on the left side, cremasteric reflex is present on both sides, but more pronounced on the left. The radial jerks upon both sides are exaggerated. The left pupil is larger than the right, and there is doubtful Argyll-Robertson pupil. Romberg is very evident. A numbness began in the glutei, and there was a doubtful sensibility of the ball of the left foot. There is no tactile anesthesia of any region, but subjective over the whole of the legs, with girdle and cuirass sensations, and there is analgesia in the region of distribution of dorsal 4th to 12th inclusive.

COURSE. It is a disease of long duration, and not directly inducing a fatality, but the disability does not allow the patient to lead an active life, and therefore he has a greater vulnerability than the normal person. There is a peculiar danger pertaining to any disease which prevents the proper emptying of the bladder, and so these patients are liable to succumb to cystitis, or pyelitis. Some of them, however, live out their allotted span of life. It is generally from one to two years before the disease becomes fully established, although there is great variation from the differences in the causative condition.

PROGNOSIS. This is good for life in the typical cases, but never for a cure.

DIAGNOSIS. This rests upon the presence of marked ataxia with a spastic form of paraplegia, and the combination of these with some sensory symptoms, and an absence of any definite and immediate atrophy.

DIFFERENTIAL DIAGNOSIS. Tabes resembles it in the symptom of ataxia, and some of the eye-symptoms might be confused, the girdle-pains, disturbances of sensibility and of the sphincters, but the ataxia of tabes occurs with a lowered or lost knee-jerk, never

with an exaggerated one. The eye-symptoms of tabes are an irregular or distorted pupil and some third-nerve palsies, but above all the Argyll-Robertson pupil, which is either absent, or very poorly marked in ataxic paraplegia. There is always some objective loss of sensibility in tabes, and it may be only subjective in ataxic paraplegia. Tabes frequently has lightning-pains and crises, while ataxic paraplegia rarely has lightning-pains and never crises. The upper limbs are almost always spastic in ataxic paraplegia, and never in tabes. Tabes is not weak in definite muscles, while the ataxic paraplegic is conspicuously weak.

Multiple Sclerosis. This is principally distinguished by the presence of symptoms wanting in the other disease. The mind is weakened, and the patient is emotional. Instead of a halting and imperfect speech, the typical multiple sclerosis patient has scanning speech, nystagmus, and intention tremor. Sensory symptoms are almost always wanting.

Spastic Paraplegia. This has weakness, spasticity, exaggerated deep reflexes, and an absence of any sensory symptoms.

Myelitis. There are two types, acute and chronic. The acute is readily distinguishable by its rapidity of onset, while the chronic form may be very similar on examination. It, however, is more limited in its distribution, and therefore the range of its symptoms will be more restricted. It tends also to run a course limited in time, while ataxic paraplegia may remit, but never recedes.

Family Ataxias. These are very similar in symptoms in the form of Marie, so far as the exaggeration of reflexes is concerned. They are, however, peculiarly familiar, are marked by a preceding mental defect, and are always earlier in life, except in the very exceptionally early cases of ataxic paraplegia. Here, however, we find in the family ataxias some nystagmus and speech defect as an early symptom.

Cerebellar Ataxia from Tumor. Here we find ataxia, but with it vertigo, vomiting, choked disc, and forced movements. While a late involvement of the pyramidal tracts may produce weakness and exaggeration of the deep reflexes, the weakness is never so marked as in ataxic paraplegia.

TREATMENT. When we make use of adjuvants, we must follow the same plan as we would follow in the treatment of a spastic paraplegia, and treat the sensory disturbances like a case of tabes. Massage is valuable in all these cases.

Electrical Treatment. In the early stages the spastic symptoms are pronounced in the lower limbs, and they may be persistent. So long as this condition persists, strong static sparks over the spine, and at the points where the nerves emerge, will be found to help the spasticity. This treatment may be modified by giving galvanism on the spine as is recommended for spastic paraplegia. When the muscles become paralyzed, faradism may

be used with more benefit, especially on the quadriceps extensor. When the sensory symptoms become evident, the static treatment of the spine should consist in drawing sparks from it for from three to five minutes; then the milder sparks from the affected parts of the body, especially over the trunks of the nerves, and then from the soles of the feet for two or three minutes. In mild involvement of the cord use galvanism with the cathode over the involved segments, and the anode over the abdomen, and an interrupted current. Another method is by including the segments between two electrodes placed upon the spine, and passing the sinusoidal current between them. When the static is used, the wave current is sometimes of benefit.

THERAPEUTICS. The best general indication is to give those drugs which have been recommended for *Tabes Dorsalis*. In the selection, however, the combined character of the sclerosis should be kept in mind, and only those drugs should be prescribed in whose pathogenesis some spastic symptom can be found. O'Connor believes that *Ustilago* may be of service, chiefly in cases affecting women. Benzoinin dinitricum has symptoms of vertigo, ocular anomalies, numbness with paralytic sensations, and spasticity.

AMYOTROPHIC LATERAL SCLEROSIS.

WASTING PALSY—SPINAL PROGRESSIVE MUSCULAR ATROPHY— CHARCOT'S DISEASE.

DEFINITION. It is a disease of the whole motor nervous system, and while it usually is located in the cervical region, it may develop a degeneration in all motor structures, from the muscle fibres to the cells in the motor part of the cortex. The lesions on the motor side are as widely distributed as those of *tabes* on the sensory, but these changes are not always consecutive. This degeneration produces a form of progressive muscular atrophy which is peculiar in showing spasticity.

THE SYMPTOMS OF A TYPICAL CASE. There is a spastic paralysis of the lower limbs, to which is added a progressive muscular atrophy in the upper part of the body.

DIFFERENTIAL DIAGNOSIS. Differentiation is from Bulbar Palsy, Tumor (cervical and spinal), Cervical Pachymeningitis, Syringomyelia, Myelitis (chronic), Spastic Paraplegia, Spinal Caries, Peripheral Neuritis, Disseminated Sclerosis, Chronic Poliomyelitis, Progressive Muscular Atrophy, other forms, Syphilis.

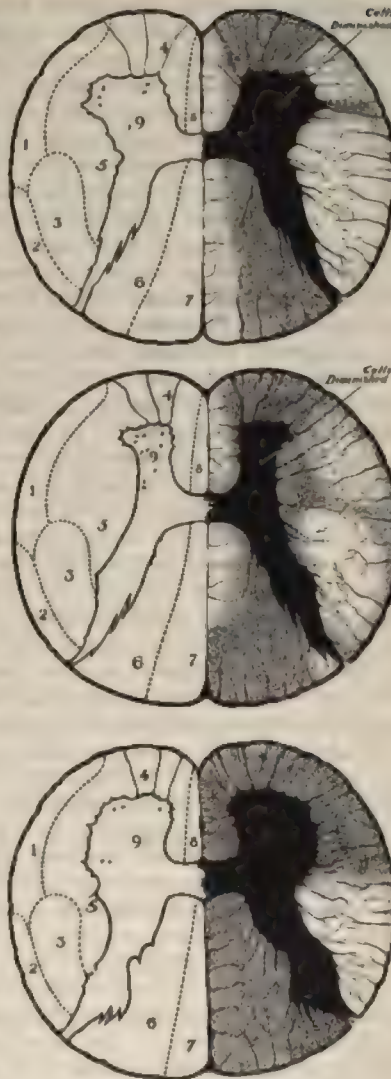
AGE. It is a disease of late middle life, and the probable limits are from twenty-five to fifty. Most cases are over thirty, yet children have occasionally been subjects of it. Here it is a secondary disease.

SEX. There is considerable evidence, but not a unanimous agreement, that females are most subject to it.

HEREDITY. This is difficult to trace, but there is in all cases a general neuropathic tendency, which manifests itself in these persons as a motor nervous system which proves itself to be too delicate to stand the strain of life.

ETIOLOGY. Granting a faulty nervous system "ab initio," the exciting cause has been assumed to have been overexertion alone, or combined with fright; exposure and deficient food; poisoning by toxic agents, as alcohol, lead, arsenic, or mercury, or the acute infections, or auto-toxins. Rheumatism, gout, diabetes may cause it, but it is not from syphilis. It may result from an extensive endarteritis, and at times from direct injuries to the back. Very apt to follow acute anterior poliomyelitis.

PATHOLOGY. A degeneration of the cells of the anterior horns is always present, and it begins in the cervical region, and in 60 per cent. of the cases is confined to them, in 30 per cent. it is confined to the legs, and in 10 per cent. involves the bulbar nuclei (Starr). If we examine the muscles we shall find that they are



FIGURES 52, 53, 54. AMYOTROPHIC LATERAL SCLEROSIS.

From below upward are sections from the lumbar, dorsal, and cervical cord. In the lumbar section we see that the degeneration is confined to the Crossed Pyramidal Tract (3). In the dorsal section the Crossed Pyramidal (3), Direct Cerebellar (2), Gowers Tract (1), and Direct Pyramidal (8), and the descending group of fibres in the posterior columns. To this is added now, for the first time, a simultaneous reduction of the motor and trophic cells of the anterior horn. In the cervical section at the top we see a more complete degeneration of these tracts, and of the cells. This explains the spastic paralysis of the legs, with a wasting paralysis of the arms.

shrunk and pale, and have lost their striation, from the fact that the fibrillae are separated one from another, and are pale. The peripheral nerves show degenerated fibres. In the cord the pyramidal, and the antero-lateral association tracts for the motor system, betray degenerative changes, which at times may be traced clear to the motor-cells of the cortex about the Rolandic fissure (motor inhibitory of the 2d, and purely motor of the 3d, layer). At other times the degeneration will die out at some one of the lower levels. The motor nuclei in the bulb may be affected. In any spot of degeneration, an invasion of new connective tissue replaces the lost nervous structures. If atrophic symptoms preponderate, we are assured that the cells of the anterior horns have suffered more than the tracts, while if spastic symptoms prevail, it is a sign of a greater involvement of the pyramidal tracts.

SYMPTOMATOLOGY. There are two classes of cases: the first where the pyramidal tracts suffer before the anterior horn cells, giving rigidity (spasticity) as the initial symptom, to be followed by atrophy; and the second, where after atrophy has developed in the upper part of the body the legs begin to show rigidity and weakness. Keeping in mind this possible alternate character of attack, we shall best understand the condition by describing as typical an attack starting in the cervical region. The patient first discovers that he is unable to perform his accustomed physical tasks without undue fatigue. He will display a slight degree of general emaciation; not specialized as in progressive muscular atrophy. At this time he may have some rather acute pains in the arms, accompanied by tingling. Examination will reveal a general exaggeration of the deep reflexes. This is termed the first stage. After several months, in one case not for two years, atrophy begins to be apparent. This begins in the muscles of the thumb, then of the interossei, and next of the little finger. In some cases the deltoid has been the first, or after the hand, and before the arm. This atrophy proceeds very slowly, fibril by fibril, but steadily progresses, until scarcely any muscular substance is left. It is preceded, and attended by fibrillation, and when it has become complete in any part the preceding rigidity is replaced by flaccidity. The order of the atrophy is the same as in progressive muscular atrophy. The loss of power will be greater during this process than the amount of atrophy would explain, since much of the weakness depends upon a degeneration of the pyramidal tracts, which in turn, even in the absence of visible post-mortem lesions, depends upon some state of impairment of vitality in the cells of the cortex. In 1881 Beevor discovered that in these cases a jaw-jerk, and in some a jaw-clonus could be obtained, and in no other cases, except one of hemiplegia. This indicates that pyramidal degeneration had reached as far upward as the motor nucleus of the 5th nerve. This is called the second

stage. When bulbar symptoms are developed the disease is said to have reached the third stage.

The above is not always the course, since a case has been reported where no atrophy was seen for two years, and then was only displayed in the tongue and lips, i.e., bulbar, nor is the atrophy always secondary to sclerosis in the pyramidal tracts, i.e., deuteropathic. A spasmodic drawing of the tongue, or stiffness of the cheeks or lips may be an initial sign (bulbar). One or two cases have been reported (Schlesinger) where an Argyll-Robertson pupil has been observed, but in general, the ocular muscles are not involved, although in late stages the upper facial may be so involved that the eyes cannot be closed. Some months usually elapse between the paralysis of the arms and the legs, but in six months the legs and arms have become so paralyzed that the patient is helpless, and long before that the contractures of the legs have become so great, that the patient has to feebly push himself along on the tips of his toes. These contractures are quite general, and result from the atrophy of some muscles, and the spastic over-contraction of their opponents. They are quite different from the contractures of progressive muscular atrophy. The fingers are bent in on the palm, the wrist is flexed, the forearm is pronated, while the elbow is semi-flexed, and the humerus is held tightly against the chest-wall. In other cases, owing to the atrophy of the interossei, the hand is clawed. In the lower limbs the rule is spasticity without much atrophy, so that in walking the leg is thrown around, scraping the toes upon the ground, but the flexion may be such as to force the patient to walk upon the tips of the toes. The head is drooped forward upon the sternum, owing to the atrophy of the muscles, and there is some degree of rigidity in maintaining the posture. When the bulbar muscles have become affected, we find paralysis of the tongue, lips, pharynx and larynx, and also the lower face, the upper face being spared, except in exceptional cases late in the disease. This wasting is preceded by fibrillation, and rigidity, and increased reflexes. In all cases, in spite of the stiffness of the limbs, the muscles are actually weak.

There are no sensory changes. Marked changes in the skin, nails, and joints do not occur.

The sphincters are not affected, except rarely, and at the very close of the disease.

Electrical reactions are mixed, as in progressive muscular atrophy. The muscles react to some degree to faradism as long as there is any muscular fibre left. To the galvanic current there is the reaction of degeneration.

The mental changes are peculiar. From the fact that the corners of the mouth hang down, the face has an appearance of deep melancholy, but actually the patients are good-natured, in spite

of a realization of their fearful condition, for the mind is unclouded and mental capacity is unimpaired. They are, however, very emotional, and without reason laugh immoderately, or burst into floods of tears.

COURSE. The duration of a typical case is from two to four years. If the attack begins in, or quickly invades the medulla, death will ensue in two to three years. If, on the contrary, it begins in the legs, the medulla may never be invaded, and the patient may live as many as thirty years.

PROGNOSIS. Unqualifiedly bad for life over four years, unless it begins in the legs. The causes of death are practically bulbar accidents. The patient may either die from lack of nourishment, from choking from food, or by some of the mucus which is more plentifully secreted than is normal, or succumb to an inspiration pneumonia.

DIAGNOSIS. In the typical cases there is atrophy of the upper extremities, combined with spastic paralysis of the lower; there is weakness, the deep reflexes are increased, but with the exception of some initial soreness and aching in the arms, there are no sensory disturbances nor disturbance of the sphincters. Before atrophy there is fibrillation, and it may be made evident by exposure to cold, or percussion of the suspected muscle. In such cases the diagnosis presents no difficulties. When it begins in the lower limbs, the only possible diagnosis for a time may be spastic paraplegia. Even when atrophy marks the rigidity, it may be discovered by the presence of the elbow and wrist jerk, by noting the contraction of the less atrophied muscles, and by what has been termed the "lead-pipe" character of the rigidity. When it begins in the bulb there may be no well-defined rigidity, or increase of reflexes; ankle-clonus may be the only indication in the limbs. There will be jaw clonus in these cases.

DIFFERENTIAL DIAGNOSIS. Bulbar palsy is held by some to be merely an upward extension of an amyotrophic lateral sclerosis, but differences may be discovered, even in cases where the bulb is invaded primarily. A true bulbar palsy does not have spinal symptoms, i.e., paraplegia, rigidities, increased knee-jerks nor ankle clonus, nor are there stiffness, cramps, or exaggerated reflexes in the muscles innervated from the bulb.

Tumor, or Pachymeningitis of the Cervical Cord. This will produce a wasting of the upper extremities, with a spasticity of the lower as the disease progresses, but its onset is marked by sensory symptoms, which are pains in the distribution of the median and ulnar nerves, and numbness down both arms, while the initial sensory disturbances of amyotrophic lateral sclerosis never are more than a painful soreness in the muscles. The onset is more rapid, and is very apt to follow an injury, and the sphincters become affected. There is objectively rigidity of the neck,

instead of the falling forward found in amyotrophic lateral sclerosis. If compression of the cord by tumor lower in the cord is sufficient to produce a spastic paraplegia, it will not produce an increase in the reflexes in the upper limbs; it will give rise to sensory symptoms, and disturbance of the sphincters, as well as atrophy.

Syringomyelia. The development is slower, and the final contractures in the hands possess a certain degree of similarity, both producing that type of deformity denominated "Claw-hand," only there are striking and essential differences. It has been stated above that amyotrophic lateral sclerosis is marked by an absence of sensory disturbances and tropho-neuroses (absolute solutions of continuity of tissue, and changes in the character of it), and by the bilateral character of the invasion of the bulb, when such extensions occur. Take up these points in succession, and observe that an absolute essential in the diagnosis of syringomyelia is the discovery of the loss of the power of perception of pain and temperature; that painless whitlow, degenerative changes in the skin and nails are usual in syringomyelia, and that when it invades the bulb its manifestations are unilateral. There is never rigidity, or increase of reflexes in the upper limbs in syringomyelia.

Chronic Myelitis may produce atrophy above, and spasticity below, if the site is sufficiently high in the spinal column, but there are bedsores and disturbed sphincters, and just at the upper margin of the atrophy there is an area of exquisite sensitiveness. Atrophy of the hand muscles is very rare.

Spastic Paraplegia cannot be diagnosed from an initial stage of amyotrophic lateral sclerosis, but it is so rare as an entity, that it should usually be considered as the preliminary stage of some other degeneration of the nervous system.

Spinal Caries if in the cervical region, produces atrophy of the hands, but there is rigidity of the spine and hyperesthesia of it. Gibbous (a projection of the hypertrophied tissue about the bones), ought to be diagnostic; the X-ray will show the condition of the bones, soon there will be in addition to spastic paraplegia the symptoms of a compression myelitis, and the history will reveal the fact that it is a secondary disease.

Multiple Neuritis is symmetrical, and always involves corresponding extremities, and usually all four of them. The onset is rapid, a few days up to a week or two; a toxic cause can generally be discovered; there are tenderness along the nerve-trunks, and marked changes in sensation, and the deep reflexes, instead of being exaggerated, are gone, or very greatly diminished.

Disseminated Sclerosis, while it has the spasticity and weakness, develops a much slighter degree of atrophy, and is sure to have some one of the following distinctive cranial symptoms:

optic atrophy of the white variety, staccato speech, or nystagmus, and intention tremor, which in itself is almost diagnostic of disseminated sclerosis.

Chronic Anterior Poliomyelitis, progressive muscular atrophy and amyotrophic lateral sclerosis have been grouped together by some authors, but chronic anterior poliomyelitis develops more rapidly; a large number of muscles, or a whole limb are atrophied at once; paralysis occurs first, and atrophy follows, while alterations in electrical response (a reduced reaction to currents of ordinary strength, and a peculiar slow wave of contraction in the muscle), occur before the atrophy has become pronounced.

Progressive Muscular Atrophy of other forms differs in that its course is slower, it attacks muscles one by one instead of whole segments, and it does not develop spasticities and increased deep reflexes. The myopathies occur in young children; the atrophies are preceded by hypertrophies, and the points of attack are the muscles of the shoulder or calf instead of the hand. They do not show fibrillation, nor do they invade the bulb.

Cervical Gliosis produces the same symptoms so far as it goes, but it is confined to a smaller area, generally a single limb, and is much slower in onset.

Syphilis may occasionally produce similar symptoms.

Multiple Arthritis produces extreme atrophy at times, but it is not in the hands, but about joints previously diseased, just as likely in the lower as in the upper limbs; the deep reflexes are not disturbed, and the condition passes away as the joints become normal.

TREATMENT. Treatment is not absolutely hopeless, and a correct hygiene adds greatly to the possible benefits to be derived. Massage must be used with great care, since an over-use is an absolute detriment. Warm baths combined with massage are beneficial for spasm, and electricity in the form of the constant current has been applied with some benefit to the spastic muscles to diminish the rigidity. For general benefit galvanism has been applied to the medulla, and the spine, and more specifically, where there is trouble in swallowing, apply one electrode anode stable, over the nucha, and the other cathode labile to the region of the neck; four to five contractions. Others have thought that good results have come from thorough local faradization of the neck and spine.

Only one result may be expected from electrical treatment, and that is a relief of the spasmodic symptoms by the same treatment as is recommended for spastic paralysis. Give a descending galvanic current along the spine for about eight minutes, the strength of current averaging from twenty to twenty-five milliamperes, using flexible hand-electrodes of about three inches in diameter. After this the anode may be removed to the abdomen, and, with

the cathode stable along various segments of the spine, a current passed for two or three minutes at each segment followed by two or three interruptions. To relieve spasmodic rigidity the anode should be placed over the contracted muscles, and here a long, narrow electrode is preferable, and the current gradually raised to its maximum of about ten milliamperes, where it should be allowed to remain for two or three minutes, and as gradually reduced. The cathode is retained on the spine. Each muscle or set of muscles should be gone over in this way. Interrupted currents over the motor-points should be avoided, except very late in the disease when paresis has become a dominant feature. Doctor Steavenson of London, recommends the electric bath. The patient sits in the bath to which is attached the cathode, the anode being placed on the spine between the shoulders. Treatments are given of ten to fifteen milliamperes for fifteen to twenty minutes. In the majority of cases the patient feels more invigorated, spasms are very much less, and locomotion is improved.

THERAPEUTICS. It will at once be seen that in a combined sclerosis of the lateral tracts which are exclusively motor, and of the cells of the anterior horns which are motor and trophic, that only such remedies will be applicable as show a spastic form of paralysis with atrophy, and with an absence of general sensory derangement.

Argentum nit. This drug has little applicability as the sensory symptoms are predominant.

Arsenicum. (Too much sensory. Clarke.) Weak paralysis with atrophy. Skin changes indicate that it might be of use in syringomyelia. (Allen.)

Cuprum met. Attention has been directed to this drug on account of its potency in subduing spasms and cramps. It, however, has extreme sensitiveness of the spine as a prominent symptom, while the disease in question has no sensory symptoms.

Lathyrus. This is recommended. Given on toxicological grounds, and while poisonings show the production of ataxia and a spastic form of paralysis, the atrophic symptom seems to be lacking. Clinical reports have not been favorable.

Phosphorus. Cramps in legs with weariness and stiffness have been used as indications. One case is said to have been greatly relieved by this drug in the 3x in combination with faradism.

Physostigma. This remedy and Belladonna are functional drugs, while Nux vomica is to be used on the same indications as Strychnia.

Pieric acid. Muscles are readily exhausted, are spastic and subjects of cramps. Many sensory symptoms are found in the pathogenesis which are not in the symptomatology of this disease. Use based upon a few clinical reports.

Plumbum. This seems to affect all parts of the cord equally,

and so sensory symptoms are prominent in the pathogenesis, but, on account of the very marked emaciation, it should be given a trial.

Strychnia. May be useful for the stage of irritation, shown by spasm, which precedes the paralysis and atrophy, indicating that destruction of cells and tracts has taken place.

Sulphur. The symptoms of weakness with spasticity, and contracting spasm of extremities, are to be found in its symptomatology.

Zinc. Too many sensory symptoms.

Not much good has been claimed from physiological remedies, and cod liver oil and tonics comprise the list in any case. Some have thought that Strychnia 1-80th to 1-30th grain daily was of value, while others absolutely bar the drug on account of the aggravation of the spasticity. Patients should be taught early the use of the stomach tube, in order to combat the emaciation and anemia resulting from difficulty of swallowing sufficient food.

COMBINED DEGENERATIONS OF THE CORD IN CONNECTION WITH ANEMIA AND THE CACHEXIAS.

DEFINITION. There is a form of sclerosis occurring in connection with the grave forms of anemia, which reminds one of the ordinary forms of combined sclerosis, except that the range of symptoms is at times wider, and at others the distribution of the symptoms does not conform to that of the more usual forms. It is also peculiar in the fact, that, at times, instead of following the anemia it precedes it.

DIFFERENTIAL DIAGNOSIS. To be differentiated from Ataxic Paraplegia.

SEX. It occurs about three times as frequently in women as in men.

AGE. It is a disease of the period between the thirtieth and fortieth years.

ETIOLOGY. Pernicious anemia is most generally the cause, but since the anemia is sometimes primary, and sometimes secondary to the cord-degeneration, it is probable that the whole condition is the result of a common intoxicant, which varies its seat of initial attack in accordance with the vulnerability of the tissues of the individual in question. It is similar to the infectious principle in chorea, where a common intoxicant is present, which is capable of producing symptoms in the joints, heart, or nervous system, or of affecting them in succession, or simultaneously. This nervous degeneration may be secondary also to other cachexias, indicating that the patient possesses a marked degree of instability of nervous tissue.

PATHOLOGY. All findings indicate that the process is inaugurated by vascular changes, which produce sclerosis as a result of hemorrhages, or by the stimulation of an exuberant growth of the interstitial tissues. It almost exclusively affects the white matter in the mid-dorsal region.

SYMPTOMATOLOGY. This is such as would be found in a case of ataxic paraplegia, plus a variable number, which can only be explained by supposing some degree of implication in the process of other parts of the cord, and also of the brain as well. At first we find a weakness of the lower limbs with spasticity, and, as would be expected, exaggeration of the deep reflexes. There is ankle-clonus, and also Babinski's toe-sign. At the same time there is marked ataxia, and, consequent upon the rigidity of the

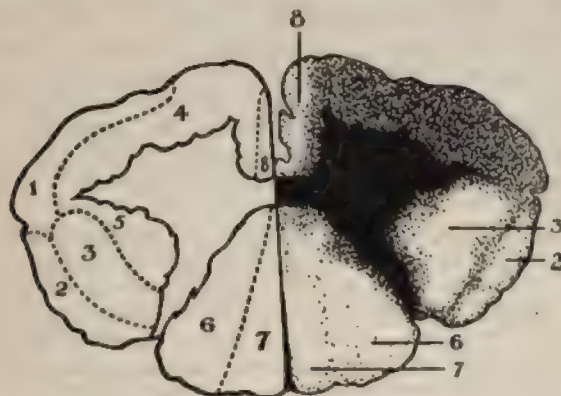


FIGURE 55. —SPINAL DEGENERATION IN CONNECTION WITH ANEMIA.

Degenerations (comparing white areas on right with scheme on left side) will be seen to affect column of Goll (7), Burdach (6), Crossed Pyramidal (3), Direct Cerebellar (2), and Direct Pyramidal (8).

limbs and the presence of ataxia, the ability to walk is very seriously compromised. There is a pronounced divergence from the symptom-complex of ataxic paraplegia in the immediate appearance of paresthesias, which are marked and persistent. Girdle-symptoms are not uncommon. Neuralgias may lead one to think of multiple neuritis. The bladder and rectum are disturbed in the line of retention.

After this condition has existed for some months, the picture is apt to change very suddenly, and the sensory symptoms become exaggerated, with an accession of lightning-pains, and an almost complete paralysis of locomotion. The knee-jerks are lost, and there is incontinence of urine and feces. The muscles become flaccid, and atrophy takes place. Instead of this stage being marked by loss of knee-jerk, it may be accompanied by an increase

in the deep reflexes, but the final stage always shows an abolition of them.

At the final stage there is swelling of the lower limbs and of the gluteal region; bedsores may appear, there is a slight degree of fever, and then the general symptoms of dissolution.

The brain is sometimes involved in the sclerotic process, and when this is a fact, there are mental irritability, and epileptiform convulsions.

Atrophy of the optic nerve has been noted in some cases.

All combinations of the above symptoms may be found, and the order above mentioned is by no means the invariable rule of its evolution.

COURSE AND PROGNOSIS. This is generally very rapid, and death usually ensues in two years, or less, but remissions do at times occur, so that cases have lasted for as much as five years. In other instances the causative anemia has been so severe, that the spinal symptoms never reach a very definite developement.

DIAGNOSIS. This rests upon the appearance of an ataxic form of paraplegia, which with brain, ocular, and sensory symptoms indicates a wider diffusion of the sclerotic process. If anemia of a pernicious grade had preceded it, the diagnosis is made with more certainty.

DIFFERENTIAL DIAGNOSIS. It is to be diagnosed from ataxic paraplegia, which it most resembles by the wider distribution of symptoms, its more rapid progress, and as a rule by its evident etiology. Sensory symptoms are earlier and more prominent.

ELECTRICAL TREATMENT. First pass a current through the section of the cord where the hemorrhage has taken place. This may be begun much sooner than in cerebral hemorrhage; a week or ten days is sufficient time to elapse. The treatment of the peripheral nerves should be the same as spinal meningitis, or myelitis. If anesthesia exist, the faradic brush may be used. The static spark has been very highly recommended, and the rules governing it should be the same as those given for spinal meningitis, or myelitis.

THERAPEUTICS. Since the degeneration is a random one, affecting any or all parts of the cord, we should treat the case like one of myelitis, with the additional element of profound anemia, which would of course be a factor in our symptomatology.

CHAPTER V

RANDOM DISEASES OF THE SPINAL CORD

MYELITIS.

DEFINITION. It is a diffuse inflammation of the spinal cord, resulting in the destruction, to a varying degree, of all the spinal elements. It varies greatly in intensity, and also in distribution. Variations in intensity cause it to be spoken of as Acute, Subacute, and Chronic, while it is called Transverse when it extends completely, or practically completely across the cord. In this case it is liable to confine itself vertically to one or two segments at the most. It is called Disseminated, or Diffuse when it has a wide vertical distribution, but is limited horizontally, so that no particular segment is extensively damaged, but many are partially destroyed. If it has extended downward from its original focus, it is spoken of as Descending, and a contrary direction gives it the name of Ascending. Locally again it may be Central, Peripheral, or Marginal, Annular, or Unilateral (rare).

THE SYMPTOMS OF A TYPICAL CASE. There is a feverish condition with a very rapid paralysis of the lower limbs, which is spastic in type, with abolition of the deep reflexes, and the incontinence of urine and feces. The muscles of the paralyzed limbs speedily waste, and there is sweating, coldness, and blueness of the affected parts. Bedsores occur upon the sacrum. Sensibility is lost in the paralyzed area, and above the insensitive parts there is a zone of increased sensibility to touch and pain.

DIFFERENTIAL DIAGNOSIS. It is to be differentiated from Spinal Meningitis, Hemorrhage into the Cord, Meningeal Spinal Hemorrhages, Spinal Hemorrhage, Multiple Neuritis, Multiple Sclerosis, Hysterical Paraplegia, Anterior Poliomyelitis, Landray's Paralysis.

FREQUENCY. It is now considered much more rare than formerly, and this arises from the fact that many diseases, formerly considered myelitis, have been differentiated, and assigned to specific categories, since they have been shown to be selective, and not general, in their destructive action, but it is one of the commonest of local diseases.

AGE. Early in adult life, twenty-five to forty-five. Children with apparently this condition are generally the victims of a

Meningo-encephalitis, while aged persons suffer from a Senile form of Paraplegia from Arterio-sclerosis.

SEX. Largely male, since they are much more subject to the vicissitudes of exertion and exposure, which are such frequent elements in the causation.

ETIOLOGY. Wet and cold, seem efficient to localize a general traumatism, or exhaustion, or alcoholism, as a myelitis. Traumatism is rarely a distinct cause, except in Caisson Disease, where the gases absorbed under pressure produce numerous capillary hemorrhages in the substance of the cord, if the person comes too quickly into the air at a normal pressure. It may be secondary to disease of the Bones. A certain proportion of cases are secondary to Spinal injuries which may leave no mark. Other cases follow Spinal Meningitis, and these are of the annular variety. The spinal cord is not as susceptible, as is the brain, to the invasion of bacteria, and to diseases arising therefrom.

Others are due to primary infection from the resulting toxins. Others result from the action of certain poisons which have a specific action on nerve elements such as Carcinoma, Tuberculosis, Syphilis, Lead, Arsenic, Alcohol, Coal Gas. For some cases, no adequate cause can be discovered.

PATHOLOGY. The essential change is an inflammation, or a necrotic degeneration from vascular obstruction. Softening is the result in either case; there is a fatty degeneration of the myelin, and resulting from this, a swelling and disintegration of the axis cylinders. The gross external appearance of the cord is not altered, but it feels softer than normal, and, on incising the dura, the contents will often well up in a semi-fluid state, and all distinction between white and gray will be found to be abolished. This general description is of course true, that there is a destruction of a whole section only in the acute transverse cases, but it is always true of the specific areas affected. No matter how extensive the lesion, some fibres will still persist. Microscopically the vessel-walls are everywhere congested, filled with leucocytes, and surrounded by those which have emigrated, and also round cells, and small nuclei. In many cases bacteria may be discovered. The intensity of the congestion is evinced by the general rupture of the vessels, and small hemorrhages are everywhere present, resulting from the destructive action of bacteria upon the coats of the arteries. Cysts may be a final result. Leucocytes are peculiarly apt to collect about the cells of the anterior horns. Cells are found to be destroyed in all their parts, cell-bodies, dendrites, and axons. From the pressure of the exudates the conduction-tracts are degenerated, and, as a result of this pressure, the myelin sheath swells, and becomes infiltrated, and the axis-cylinder degenerates in turn. By this cutting off of conduction-tracts, cerebral control is lost, and the local degeneration destroys the efficiency

of the segments involved in their rôle of reflex arcs, or secondary or automatic centers.

After this solution of the normal functioning structures of the cord, a sclerosis occurs, which replaces none of them in a proper sense, but substitutes for them an overgrowth of neuroglia-tissue, which is practically the formation of a scar. This is the result of an irritative reaction, which sets up karyokinesis of the neuroglia cells. The bacteria usually found are *Streptococcus*, White *Staphylococcus*, Yellow *Staphylococcus*, *Diplococcus* of a special kind, which has not yet been so accurately differentiated as to be termed the *Diplococcus* of Myelitis. There are many cases where no bacteria can be found, and it is a question whether they have been on the field, and have disappeared, or were never present. It is to be remembered that here, as elsewhere, nerve-elements once destroyed are never replaced.

Transverse myelitis in a large way, and other forms as surely, but less generally, cut off fibres from their distant nutrient cells, as well as injure cells at the site of the lesion. After a fibre is thus cut off, a degeneration is set up in the axis-cylinder, which extends in a direction away from the cell-body. Therefore motor degeneration is descending, while sensory is ascending, and the distribution of both these degenerations is greatest at the site of the lesion. We

shall therefore find all the motor fibres degenerated below the lesion, and since the association tracts, and the comma tract of Schultze lying in the column of Burdach, and the septo-marginal tract lying in Goll, are also motor, they will also be found to be degenerate. In the lowest part of the cord the sensory fibres are all comprised in the columns of Goll and Burdach, although the tract of Gowers is beginning. The direct cerebellar begins at the first lumbar. Sensory degeneration extends upward, and there-

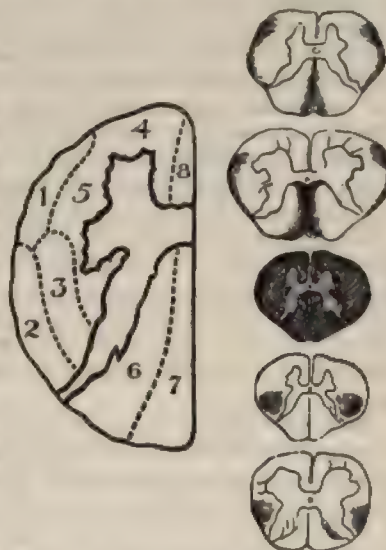


FIGURE 56.—COMPLETE TRANSVERSE MYELITIS OF THE DORSAL REGION.

Section of the Cervical Cord is at the top, of the Sacral at the bottom. Referring to the schematic diagram, it will be seen that the destruction of an entire section of the cord produces an upward degeneration of the sensory Columns of Goll (7), Direct Cerebellar (2), and Gowers (1). It produces a downward motor degeneration, i.e., Crossed Pyramidal Tract (3). Reference to the schematic half will show the location of the affected tracts.

fore the higher the site of a lesion in the cord, the wider will be the resulting area of degeneration.

Secondary degeneration results from any sort of lesion of the substance of the cord, as we have seen in a Myelitis of the primary variety, and also from secondary forms resulting from tumor, caries of the spine, fractures of the vertebrae, hemorrhages either within or without the cord, wounds of the cord. It begins within twenty-four hours of an injury, and is an active process for from six to eight weeks.

SYMPTOMATOLOGY. This is of three classes, Paraplegic, Segmental, and Root, and their relative importance will vary according to the horizontal and vertical extent of the lesion, according to the particular segments involved, and to the degree to which the primary disease has involved the nerve-roots. The gravity of the resulting damage depends largely upon its site. Paraplegic symptoms are such paralyses and motor phenomena as are developed by the interference with the conduction-tracts.

Segmental symptoms are such as arise from the destruction of motor, sensory, and trophic (?) cells in the affected segments. Segmental lesions produce atrophic forms of paralysis in muscles which have been previously furnished with their nerve-supply by the cells of the segment in question. We shall also find sensory disturbances in the areas of skin innervated from these segments.

Reflexes will also be abolished, if their arcs are involved in these segmental softenings. It is a point of some diagnostic importance whether the motor or sensory symptoms are accompanied from the first by a paraplegia, or whether that is a later development. If the paraplegia is an early symptom, we have good reason for believing that the cord is the primary seat of injury, but if the paraplegia comes on later, it must be considered that the injury to the cord is secondary to some other process, and that the cord is only injured by compression.

Root-symptoms are these. Pain is the most constant evident factor, and is usually sharp, neuralgic in character, and sometimes tender points develop. It may be intermittent, or constant, and, if in the limbs, is apt to be referred to the joints. This is at times the only symptom. There is usually an intense hyperemia of the skin, and yet, in spite of this general increase of sensitiveness, we are apt, in the later stages, to find areas of anesthesia to which pain is referred. Such a condition is called *anesthesia dolorosa*. All the above-mentioned changes of sensation are strictly limited to the areas which derive their fibres from the segments of the cord which are the seat of the disease. Irritation of the motor nerve-roots may also cause painful contractures of the muscles which they innervate, but this symptom is more rare than a gradual loss of power from interruption of the fibres in their course.

The interruption of motor fibres causes an increasing degree of muscular weakness accompanied by wasting.

The rapidity of this atrophy varies considerably, and with it the degree of change in the electrical reactions. If the atrophic paralysis is slow, there will be found to be merely a progressive decrease in both faradic and galvanic irritability; if it is more rapid, there is often the reaction of degeneration, and, at times, what is known as the mixed form of reaction, on account of the partial character of the damage of the fibres to the muscles; and a definition of this form is this: the reaction in the nerve is normal, and altered only in the muscles.

The most common form of the disease is a Transverse Myelitis of the Dorsal Region, and the next is Transverse Lumbar.

Transverse Dorsal Myelitis. This is the most common type and location. In this variety of myelitis, there is a minimum of segmental symptoms, since the segments at this level supply only the muscles of the abdomen, and the lower thorax, and losses must be very complete to be conspicuous. We are mainly impressed by the following: Loss of voluntary motion in the lower limbs; loss of sensation in all parts below the level of the lesion (usually accompanied by a zone of hyperesthesia, about two inches in width, just above it); loss of control over micturition and defecation. There will be a variation in the degree of completeness of such losses proportioned to the completeness of the lesion. When the lesion is incomplete, there may be a disproportion between losses of one variety and those of another, but it may be broadly affirmed, that with a variable degree of injury to the tracts of the cord, the motor palsy will be greater than the loss of sensation, or of control over the functions of the bowels and bladder. Starr says that at times the sensory change is an erroneous localization of sensory stimuli.

The paralysis of the lower limbs (paraplegia) is at first a simple loss of power, which develops into a paralysis of the spastic type; i.e., the limbs grow more and more rigid, and draw up spontaneously, or when manipulated, and eventually become more or less completely fixed in some degree of flexion. This is due to two causes: the first is a loss of inhibition over the unrestrained reaction of the motor cells of the anterior horn in answer to the peripheral stimuli, which are always inciting them to reflex action, and which in health is regulated by the corrective influences sent down in the motor tracts, which by this disease have been cut off; secondly, from the fact that from the lack of nutritive stimuli, exerted through these same motor tracts, the motor cells are in a state of anemic irritability. Knee-jerks are exaggerated, there are ankle-clonus, and the Babinski toe-sign. For reasons which are still under discussion, a complete division of the motor tracts by myelitis produces a picture differing from that which we might

expect. In such cases the knee-jerks are not exaggerated, but are abolished; perhaps because muscular tonus, which in the incomplete lesion was still in some measure distributed to the motor cells of the anterior horns, is now completely cut off. The converse of this, viz., that, if the knee-jerks are absent, we have proof that the existing myelitis is complete, is not always a truth. In the early stages of a myelitis, such a total loss of knee-jerk may be the temporary result of the shock of the invasion. The muscles retain their volume, and normal electrical excitability, even after paralysis has existed for a long time. There is anesthesia, and girdle-pains about the waist may be present, and, if the posterior nerve-roots are especially attacked, there will be severe lancinating pains in the area of the distribution of these nerves (root-pains).

Retention of urine is usually an early symptom in severe cases, and must be relieved by the use of the catheter, but, as the case goes on, this condition may be replaced by an incontinence, due to paralysis of the sphincter. Priapism is often present, it is common in cervical, frequent in dorsal, but absent in lumbar localization, and impotence is also found in some cases.

The bowel shows the same aberration from the normal, in that a primary retention of the feces is likely to be followed by involuntary, and unconscious discharges. In lesions above the mid-dorsal region we find much gaseous distention of the bowels, and the bladder has been known to rupture. Bedsores are common, and develop with great rapidity, beginning with a patch of erythema, which assumes a darker color in the middle, followed by blebs filled with fluid, which rapidly break down, and reveal a deep-seated and extensive slough, which often reaches down to the bone. They are caused by the disorganization of the trophic elements in the cord, but they may be avoided, in some cases, by surgical cleanliness, and certainly are favored by the irritation and moisture resulting from imperfect care of the excretions. Vasomotor and secretory disturbances are present; edema, arthropathies, anidrosis, etc.

Lumbar Myelitis affects segments which are motor, sensory, and trophic to the lower limbs, and leave the power and sensation of the trunk unaffected, or, at the most, impairing it at its lowest levels. There will be paralysis of the lower limbs, which will be flaccid in type, that is, the legs will hang powerless, with the knee-jerks abolished, as well as all other deep reflexes; the skin reflexes are also lost, and, since the nourishment of the tissues depends in some way upon the integrity of the anterior portion of the cord, there will be wasting. There may be a lack of completeness in the lesion, and if the upper part of the lumbar portion of the cord is healthy, the knee-jerk will be spared, or even exaggerated as a phenomenon of irritation, since the ilio-inguinal, the

crural, and the obturator nerves are not involved in the area of destruction. There will be anesthesia of the legs, and perhaps a slight involvement of the lower abdomen. There will be no girdle-pains in this case, but instead, there will be radiating pains in the distribution of the nerves of the extremities. The paralysis of the bladder and rectum will be very pronounced. There is a group of cases where the paralysis gradually appears, first in one, and then in the other limb, and finally develops into a typical paraplegia. These cases are called Chronic Transverse Myelitis, but will probably develop into a Multiple Sclerosis.

Sacral Myelitis produces these same anesthetics, overflow, incontinence of the bladder, and no power in the rectal sphincter. The over-distention of the bladder favors the development of cystitis. Being below the origin of the nerves to the lower extremities, there is no paralysis of the lower limbs, if the myelitis is strictly limited to these segments.

Myelitis of the *Conus terminale* gives rise to many of the symptoms which we should naturally ascribe to a lesion of the sacral portion of the cord, since it is composed of the mass of nerves from these segments, just before they are spread out in the cauda equina. There is paralysis of the bladder and rectum, the patient is impotent, there is anesthesia of the anal, scrotal, and perineal regions, of the penis, of the upper and inner surface of the thighs, and sometimes a degenerative paralysis of the sciatic region.

Cervical Myelitis. The symptoms here will differ, according to the involvement or escape of the cervical enlargement. When this is involved, the arms will naturally show an atrophic, flaccid paralysis, while the legs will be spastic, since the cutting off of the motor tracts anywhere above a reflex center takes away inhibitory control, but a segmental loss results in atrophic paralysis. There will be anesthesia of both arms, and of all parts below the lesion, as in all other localizations, but not always complete, since some fibres may remain intact in almost every localized inflammation. This lack of sense-perception comprises all varieties, tactile, pain, and temperature. Respiration may be imperfect, from paralysis of the diaphragm through the phrenic nerve. There may be pupillary symptoms, since the movements of the pupil are strongly influenced by the condition of the lowest cervical sympathetic ganglion, which gains its fibres from this, and the upper dorsal segment. The pupil is usually dilated, and in disseminated cases optic atrophy is common. Dysphagia, hiccough, variations in pulse (slow, or fast, or irregular), and hyperpyrexia.

If the lesion is above the cervical enlargement, the direct motor and trophic innervation of the arms and the intercostals escapes; that is: it is no longer a segmental loss, and so there is no atrophy, but the arms are the seat of a spastic paralysis, while respiration is still affected by the paralysis of the diaphragm through the

roots of origin of the pneumogastric, and, if the lesion is still higher, the medulla is involved.

Incomplete Transverse Myelitis is not uncommon, and then the picture is confusing from the incompleteness of some, and the absence of other symptoms. Paralyzes may not be complete; rather those degrees of motor loss which are termed "Pareses." Instead of anesthesia, we find hypesthesia, which is a diminution, instead of an actual loss of sense-perception. In other cases, the sensory loss will be confined to one side of the body, and sometimes the lesion is so strictly confined to one side of the body that we find a perfect picture of what is termed "Brown-Sequard Paralysis," which is this: on the side of the lesion is a motor palsy, while the anesthesia is on the other, yet with this reservation, that muscle-sense is lost on the paralyzed, and preserved on the opposite side. This sensory phenomenon is supposed to owe its existence to the fact, that all sense-perceptions, except those from muscles, and tendons, and joint surfaces, are conveyed in tracts, which cross to the opposite side of the cord, immediately after their entrance. Mott and Turner (Oppenheim) have not been able to verify this histologically, but it seems to have been clinically established. On the paralyzed side, there seems to be some sensory exaltation, especially to pain. Temperature is generally raised from 1-2 to 1°. The functions of the bladder and rectum are usually, but not always, disturbed. On the paralyzed side after a little interval of loss, the knee-jerks are exalted. On the paralyzed side there is a half-girdle of hyperesthesia, and pain, while on the other side there is a half-girdle of anesthesia with a border of hyperesthesia above it.

It is a rule to be remembered, that all signs of a transverse break in conduction are present, if only part of them are suggested. The vertical extent of the process may be very considerable, e.g., the whole dorsal, or parts of the dorsal and lumbar may be involved, and, of course, such an extent of lesion may modify the symptomatology.

DISSEMINATED MYELITIS. This is a form of myelitis where patches and tracts of inflammation are scattered throughout the cord, and even involve the medulla, and the brain. It is evident that the lesions may be so distributed, that unitedly they might constitute a complete cross-section, and the symptoms, so far as they relate to conduction, will be similar to the transverse form, but diagnostically they will show very few completely marked segmental symptoms. The causation is an infection or intoxication, from whose general diffusion small foci of inflammation are set up at numerous points.

Many cases are atypical, and often symptoms flowing from implication of the brain, or medulla, will precede those from the spinal cord. There may be initially, the paralysis of an arm, or

of a cranial nerve, or the onset may be like an apoplexy. The involvement of the brain may cause symptoms like those of chorea, but, as in other forms of myelitis, a paraplegia is apt to be the prominent symptom. Optic neuritis has been known to occur. We find intention tremor and ataxia in both, and it is said that some cases develop into a multiple sclerosis, but such a statement is a confusion of terms.

COMPRESSION MYELITIS.

DEFINITION. Certain cases of myelitis arise from no disease of the cord itself, but are the results of compression by some condition arising in the structures outside of it.

ETIOLOGY. A very common cause is caries of the vertebrae, or tuberculous disease of them, or traumatism which dislocates or crushes them. The usual result in these cases is for a collection of pus to invade the spinal cavity, and to compress its contents. Without this purulent result the bones may break down and assume new positions, which encroach upon the cavity of the column. In case of traumatism there may be a subluxation of the bones, or parts may be driven into the canal (laceration of the cord is excepted in this discussion), or the intervertebral cartilage may be displaced inward, or the resulting hemorrhage from the accident may form an encroaching mass. Tumors may arise from the bones, or from the periosteum, or from the membranes. Aneurism of the descending aorta may erode the bones of the lower half of the dorsal region, and allow them to sink inward, and a cancerous growth of the posterior mediastinum has infiltrated the tissues, and invaded the cavity in a case seen by the author. Echinococcus cysts have also been the agent for the production of pressure. All these conditions will not infallibly produce a myelitis, but are sufficient causes if the pressure is sufficiently severe and of long duration. The spine may be injured or seriously diseased without a compression myelitis being the necessary result.

SYMPTOMATOLOGY. This must conform to those just given, to enable us to state that a myelitis has resulted. Root-pains are common in both conditions, and if they occur before the paraplegia, we must consider that the causative condition is without the cord.

COURSE, PROGNOSIS, ETC., OF ALL FORMS OF MYELITIS.

COURSE. The onset may be very acute, needing the passage of only a few hours to develop the complete symptom complex. Such a case is sometimes termed Myelomalacia, or Spinal Softening. Usually from one to ten days are consumed in the process,

and chills and fever show that there is an inflammatory process from some source of infection. Starr quotes a case of disseminated myelitis which took twelve days to develop the paraplegia, and one of the transverse type in the lumbar region where it was complete within four hours of the onset. With, or before the chill there is a feeling of numbness in the legs, followed by an exaggeration of sensibility which is later replaced by an abolition, or reduction of it. The invasion period is sometimes marked by pain, but it is never a pronounced feature. There is next a weakness in the lower limbs, which speedily becomes a paralysis, but some cases are very insidious, and in non-fatal cases the development of the disease is very slow. In some cases there may be only a weakness of the legs, always with some stiffness, and the sensory changes are equally slight. However slow the onset, disseminated myelitis will finally display to some extent the more definite symptoms of a localized myelitis.

PROGNOSIS. Typical cases are usually fatal, but a difference in etiology produces a difference in symptoms, and degree of severity, and probability of fatality. Some result in a myelitis which tends to recovery, while others result in chronic forms, and still others, to a type marked by remissions and relapses. Generally the infectious cases, such as result from variola, typhoid, erysipelas, influenza, etc., and which resemble an acute ataxia, tend to recover. Gonorrheal cases almost universally do well, as do those arising from malarial poisoning, but these latter are marked by a strong tendency to remittency. Syphilitic meningo-myelitis is not necessarily fatal in its outcome. Some cases complicated by meningitis, and neuritis, have recovered. Acute, or incomplete cases, are more favorable in prognosis than those of insidious onset, or the completely transverse. The slender, and the aged, bear the disease better than the robust and the youthful. The return of sensation to an anesthetic part is a sign of recovery, while an extension of this loss is of bad omen. Bedsores, and complete paralysis of the bladder and rectum are bad signs. The longer continued the case, the worse the prognosis, and death usually results from bedsores, or cystitis. An attack coming on in the puerperium, or in the course of tuberculosis has a bad prognosis. However great the degree of recovery, some amount of spastic paraplegia will remain in the lower limbs.

DIAGNOSIS. This rests upon the fact that suddenly, or in a few days, a person develops a paraplegia, with exaggeration of the deep reflexes, the only exception being, when the segments containing the reflex arc are destroyed, and then, in that particular reflex only, we find complete loss. There are segmental symptoms, and to these are added root-symptoms, if the cause of the myelitis originates in the meninges. The functions of the rectum and the bladder are disturbed; there is atrophy of the lower limbs,

and blueness on the surface, and profuse sweat. Bedsores almost always occur. Whether the lesion in the cord be complete or incomplete, there is a persisting plantar reflex, of the extensor type.

DIAGNOSIS OF SITE. The upper level of the anesthesia affords the best means of estimating which segment is the uppermost one affected. This border is apt to be definite in outline, but if above it, as is common, we find a zone, an inch or an inch and a half in width, we may be sure the lesion is not higher than the segment just below the one which gives off the fibres to the hyperesthetic zone, since this is a symptom of irritation, and not of destruction. It is not so easy to locate the damage by the impairment of muscles, since each muscle, though mainly innervated from a definite segment, is partially innervated by others in the neighborhood. Determination of the lowest border of the injury is less definite, and we can only say with certainty, that if the bladder and rectum are still tonic, the lesion is above the lumbar enlargement.

DIFFERENTIAL DIAGNOSIS. Spinal Meningitis is usually secondary to meningitis of some other part of the cerebro-spinal axis. When the cerebrum is primarily the seat of the disorder, the spine may not attract notice, but if the spinal meninges are the primary location, we shall find more pain, rigidity, convulsions, and fever, than are usually found in a myelitis.

Hemorrhage into the Cord is, in its inception, very different from myelitis. The patient gets a paraplegia, but not in a few days or hours: on the contrary, he is apt to drop as if struck, and a definite spot in the spine is the seat of an acute pain. In myelitis, pain is not a pronounced symptom, and, if present, is not necessarily confined to the spine. After a few days the symptoms are similar, since the hemorrhage sets up acutely the same necrosis in the substance of the cord, which occurs more slowly in a typical myelitis.

Spinal Meningeal, and Spinal Hemorrhage, differ at their onset from myelitis in possessing the same instantaneousness of attack that has been spoken of under the preceding heading, but more exactly speaking, rapidity of onset differentiates Hemorrhage into the Cord, and Myelitis. In Spinal Meningeal Hemorrhage there is likely to be a pain in the back in a definite spot, and root-pains are noticeable, since primarily from the meningeal site, or from a secondary implication of the meninges, the roots are involved. From this factor we get more initial rigidity of the muscles of the back than in myelitis. The final result of meningeal hemorrhage is the production of a compression myelitis.

Multiple Neuritis. This typically involves all four extremities, and if the face is affected, it is surely not a myelitis. It is a peripheral disease, and if it involves the lower limbs alone, it

is distinctive, in that it leaves the muscles near the trunk free, or it is true up to a period of very complete developement; in myelitis the whole musculature of the limbs affected is paralyzed together. The bladder and the rectum are practically never involved in multiple neuritis. The muscles are tender in neuritis, and not in myelitis.

Multiple Sclerosis. Owing to the similarity in the site and distribution of the lesions it is not surprising that there should be points of similarity in the symptoms, although multiple sclerosis is degenerative, while myelitis is inflammatory. The onset of myelitis is abrupt, and may be marked by chills and fever, while that of multiple sclerosis is gradual, without such accompaniments. This difference of onset, and the rate of subsequent developement, may be the only distinguishing mark in atypical cases. Both have an ataxia, and paraplegia, and both a tremor, but it is very rare that it is of the intention variety in myelitis. Myelitis never has the staccato speech, nor the nystagmus which are symptoms in typical cases of multiple sclerosis.

Hysterical Paraplegia. This occurs almost always in the female, while in youth, and there has been a recent emotional strain of a pronounced character, or of less severity, but of longer duration. The paralysis of hysteria is apt to be a monoplegia, while in myelitis, it is almost a rule, that it is a paraplegia, and the resulting rigidities are not, in hysteria, the obvious result of peripheral stimulation, but a response to an exteriorized mental picture of rigidity. This is shown by the fact, that if the paralyzed limb is raised, and the sustaining power suddenly but unobtrusively withdrawn, the limb will not fall, but will gradually sink by hitches. A specific atrophy is rare, bedsores almost unknown, and incontinence is absent; the vesical fault, if present, being retention.

Acute Non-progressive Disseminated Encephalo-myelitis. This is a poorly defined form of disseminated myelitis, which is not so fatal as the acute form of disseminated myelitis, and has been termed by von Leyden, "Acute Central Ataxia." It is apt to occur in the course of, or soon after the acute infections, small-pox, typhoid, and variola, and so is most often seen in children and young persons. On account of the severity of the initial disease, the early symptoms are apt to be considered to be unusual symptoms of the primary disease, but, as it passes away, the patient is found to present the following symptoms: loss of speech; loss of power in the limbs; changes in tendon reflexes. Ataxia may be the first symptom, or it may follow unconsciousness, restlessness, or delusions. In the ataxic state there may be intention tremors of the limbs and head, disorders of speech, especially of the scanning variety, but there may be dysarthria. Nystagmus appears to be uncommon. Aphasia, or optic neuritis,

sometimes occur in the earliest stages. Death is rare; a few cases recover quickly, and in others some symptoms, especially speech disturbance, persist. There are many points indicating that it should develop into multiple sclerosis, but German authorities deny that this does occur.

TREATMENT. The treatment of this condition is not to be approached with a great deal of optimism, since it is not very efficacious as a rule. Most drugs have been found to be useless.

From the very first, absolute rest in bed, even when voiding secretions. Cupping has been recommended for the early stages, when marked by pain, or the same effect has been obtained by ice-bags, hot fomentations, hot sand-bags, or salt-bags. The application of heat may be more pleasant if bran-bags are used, but they must be frequently changed. The wide diffusion of the electric current makes it possible to make use of the various kinds of electrical substitutes for these before-mentioned appliances, and they have the great advantage of being light, and manageable, and the greater merit of needing no renewal. If the case follows an infection or an intoxication, free sweating should be maintained, to aid in eliminating the poison. Vesication has also been used, but it should not be over anesthetic zones. Iodine is a good revulsive in these cases. We may obtain the two-fold advantage of free diaphoresis, and local heat, by the use of the wet pack in the early stages. Hot baths should be avoided, and warm baths only after the disease has ceased to progress (Oppenheim). ("Either cold, 60°, or hot, 100°, are bad." Starr.) Experience has shown that cold applications are more generally valuable and grateful than hot ones, except in those cases which have arisen from exposure to cold. The bowels should be moved every other day, and the catheter should be used with extreme attention to asepsis, and the scrotum should be carefully protected with cotton, to avoid the retention of dampness in its folds. The possibility of bedsores should be ever before the attendants, and their appearance should be combatted by cleanliness, to a surgical degree, careful drying of the parts from body-sweats and the secretions, dusting with zinc, or other powder, and an avoidance of wrinkles in the bed or body-clothing. Greatest in value of these, is the frequent change of position, and most efficient of all is the use of a water-bed. When the skin becomes dark, and a slough seems imminent, bathing with cologne, or alcohol, weak solutions of tannin, or alum, temporary applications of dry heat, or positive galvanism may abort it. If it is possible, the continuous bath may be employed. In more chronic stages a sojourn at some warm springs may be of advantage, but water at a high temperature, and sea-bathing should be avoided. If cystitis appears, the bladder should be washed out once or twice daily with a solution of Permanganate of Potassium in a 1-8th to 4% solution, or

Boric acid 10%, at 98°, two or three times a day, Salol grs. 5, or Urotropin 10 grs. every four hours by mouth. Other drugs may of course be applicable. The most perfect action of the bowels, the kidneys, and the skin, is essential, not only for general reasons, but also because their perfect function seems to actually drain the spine.

Spontaneous contractures may be unavoidable, but they should be combatted by massage and electricity (neither in the early stages of the disease), and passive movements should also be used. As strength returns in the limbs, the patient should endeavor by concentration of mind to produce motions with the affected limbs.

Electrical Treatment. During the acute stage of the disease electricity has no place, but is valuable in the treatment of the resulting paralyses, anesthasias, or contractures. While it has been advised that the cathode of the galvanic current should be placed over the segments involved in the disease, and the anode upon the abdomen, it is a rather better plan to place the anode upon any part paralyzed, and so complete the circuit. In using this current no interruption should be made until all signs of inflammation have disappeared, and the current at the beginning should be just sufficient to redden the skin at the cathode, and should not be passed for more than five, or at the outside, ten minutes at a sitting. When the condition has become chronic, and the patient has borne without destruction or aggravation these moderate currents, they should be increased up to the point of vigorous contraction of the muscles involved, and for that purpose the polarity should be reversed, having the cathode over the motor-points. The sphincters of the rectum and bladder are incompetent to some degree in all cases of myelitis, and should be the subject of treatment, as soon as it is instituted for any paralytic loss. This is best treated by placing the anode upon the back just at the lower border of the shoulder blades, and the small cathode should be placed upon the anus, and the interrupted current should be passed between the two. The urethra may be similarly treated by introducing a steel cathode into the urethra, and passing a mild current between the two, but care must be exercised that no electrolysis is produced. Paralysis of the bowels may be favorably affected by putting the negative electrode upon the anus, and the positive swept about the bowels in the direction of the large intestine. The effect is sometimes greatly increased by giving a high enema of normal salt solution, and introducing a hollow anodal electrode into the rectum, and using the cathode upon the abdomen. After the muscles have developed an easy response to the galvanic stimulation, faradism may be substituted with probably equally good results, and is often more feasible in cases where patients are not possessed of ample means. The faradic is also the best for treating the areas of

anesthesia, where it is applied by means of the one pole being placed upon any indifferent point, and the other being applied to any anesthetic spots by means of a wire-brush electrode. The abdomen may be treated with the static spray in cases of intestinal paralysis, and the same treatment has been applied to the perineum for incontinence. The high-frequency effluve, administered with a glass rectal electrode, has been quite successful in my hands in the treatment of fecal incontinence. The contractures have been benefitted by continuous galvanic currents to the spine, including the diseased segments between the poles, terminating the seance with short, sharp interruptions, and then applications to the muscles.

THERAPEUTICS. It must be recognized that the condition is, at first, an acute inflammation of nervous tissue, and that is the point in the disease in which our therapeutic measures are most likely to be of advantage. In addressing ourselves to this condition, experience has taught that the most success attends our efforts when they are directed along causal indications. If the cause has been an injury, we should give *Arnica*, *Aconite*, or *Bellis perennis*, *Hypericum*, and sometimes *Belladonna*. If it follows closely upon exposure to cold or wet, *Dulcamara* or *Rhus tox*. While we are giving these remedies we should not neglect to remove any acute inflammation, or source of infection, to which the myelitis may be secondary. At times it is difficult to form any opinion of the etiology, and, in such case, *Mercurius* in the 3x has been reported to have been very efficacious. It certainly exhibits in its symptomatology a progressive paralysis of the extremities, of the bladder, and rectum, with pains in the spine which are aggravated by pressure, and anesthesia in the limbs, and there is a tendency to painful spasms of the muscles. If there is a possible syphilitic causation, a mixed treatment should be energetically inaugurated, or *Syphilinum*, in a high potency, given. If the invasion has the rare localization of the cervical portion of the cord it will be characterized by a violent priapism, and in this case *Pieric acid* is the best remedy which we have for the condition. Remedies of a more general character are *Cuprum arsenicosum*, which has the symptoms of myelitis, with the spasmodic action of the limbs very strongly marked; *Arsenicum*, which is to be thought of for organic cases where the cord symptoms alone are prominent, while *Oxalic acid* is to be used in its place where the meninges are also implicated. This will be shown by the presence of root-pains, and tenderness in the back. This remedy, however, seems to have a very wide application to all degenerative and inflammatory conditions in the spine. *Nux vomica* is also a remedy indicated where there are paralysis, sensory changes in the limbs, and root and girdle-pains, showing that the meninges in this case also are affected. The applicability of *Rhus* to organic

conditions is very doubtful, since the pains which have been described as root-pains seem rather more probably to be located in the muscles than in the nervous structures. *Artemesia abrotanum* has aching in the back, with numbness and paralysis of the limbs, and has some clinical evidences in its favor. Phosphide of Zinc should be thought of where the spinal pain is excessive. In organic conditions which seem to be in a condition of stasis, *Nux vomica*, *Ignatia*, and *Strychnia* in all potencies, and in the dosage seeming to be applicable to the given case, are the best remedies. *Strychnia phos.* 3x has often been of advantage. Other remedies to be considered are *Bryonia*, *Cicuta*, *Agaricus*, *Gelsemium*, *Phosphorus*, *Physostigma*.

Physiological Remedies are mainly the use of Bromides for the cramps in the muscles, and sometimes Morphine and Hyoscyne are used for the same purpose, but the Bromides are the better of the three. For cases arising from infectious causes, use is made of the Salicylates, and mixed treatment if it is the result of syphilis. Mercury has been used for cases of all descriptions, on account of its action upon interstitial nervous tissues, but its value is considered to be very problematical. Quinine and Arsenic are advised for cases arising from malaria. Ergot has been used extensively for all spinal conditions, but has not proved to be of as much value in myelitis as in some other diseases. For the resulting paralysis *Strychnia* is almost universally used, either hypodermatically, or by the mouth.

SPINAL TUMOR.

DEFINITION. The definition must include new-growths in all parts of the spinal canal, whether arising from the internal surface of the vertebrae, the dura, and the pial membrane, as well as from the structures of the cord itself, since a tumor situated in any of these structures will so soon set up symptoms pointing to an implication of all of them that anything more than a vertical localization is impossible.

SYMPTOMS OF A TYPICAL CASE. A male, from thirty to fifty years of age, gradually develops pain in a definite portion of the spine (in the dorsal region, or in the cauda equina), which is at first a unilateral, intercostal neuralgia, which later becomes bilateral. Next there are girdle sensations in addition, which are in turn followed by pain in certain areas of the body which are innervated from definite segments of the cord. These segments are found to correspond with portions of the spinal column which are locally painful. These pains increase through months, and sometimes a year or two, and the reflexes below this segment are increased, but are soon lost if a reflex arc is the focus of disease. Paraplegia

next ensues, followed by a loss in sensibility below the seat of the lesion. Lastly, all reflexes below the lesion are lost, there is wasting of the lower parts of the body, which may be intense enough to produce bedsores, and more rarely there is spinal deformity, and pressure over the spine at the site of the tumor may set up a tetanoid condition.

DIFFERENTIAL DIAGNOSIS. This is from Caries of the Spine, Carcinoma of the Vertebrae, Pachymeningitis Cervicalis Hypertrophica, Transverse Myelitis, Aneurism of the Aorta, and Sciatica.

AGE. Thirty to fifty. Under thirty-five years of age the growth is usually tuberculous.

SEX. Males most often.

HEREDITY. The multiple tumors are generally hereditary.

ETIOLOGY. Syphilis, tuberculosis, and the carcinomatous cachexia are the commonest causes, and traumatism is a common exciting cause. Parasites occasionally.

PATHOLOGY. Lipomata, and echinococcus cysts are always extradural, and the tumors springing from the nerve-roots may also be. Internal to the dura are myomata, sarcomata, psammomata, syphilomata, tubercle and fibromata. Sarcoma may develop as multiple tumors scattered over the whole extent of the meninges, and it may to some extent replace the tissues of the cord itself, but gliomata are as a rule most wide in their diffusion. Sarcomata are not only found in the cord, but may also simultaneously appear in the spinal nerves and the brain. Neuromata and sarcomata are found on the spinal roots. Echinococci and cysticerci are found, the former being seated upon the vertebrae, while the latter are on the membranes. Intra-dural tumors are generally small, oval, or cylindrical, with the long axis corresponding to that of the spinal canal, and are liable to attain their greatest size in the site of the lumbo-sacral region, and that of the cauda equina. They are often limited to one side, and therefore a Brown-Sequard type of paralysis may be the initial form, followed later by a paraplegia. The extra-dural tumors do not tend to invade the spinal canal by destroying the dura, and it is rare that such as are intra-dural are found to pierce the pia and invade the substance of the cord. The size of these growths varies from that of a pea to that of a body of an inch or two in length, and an inch in thickness. The favorite sites of location are the upper and lower dorsal region, and next among the fibres of the cauda equina. Tumors of the meninges are small, but may follow the meninges for an inch or two, or form a sheath about the cord. Most spinal tumors are of very slow growth, but syphilomata are always of rapid growth, and tubercle is generally so, and may even surpass syphilomata in that respect. While compression of the cord is the usual result, and this may reduce it to one-fourth of its bulk, any actual invasion of its tissues is rare, and its structure is rarely

changed, but inflammation may ensue with softening. Glioma and sarcoma are the commonest, fibroma and myxoma next, and then gumma and tubercle. Tumors may form cavities in the cord.

SYMPTOMATOLOGY. The disease is first manifested by the occurrence of a unilateral intercostal neuralgia (root-pain) which soon becomes bilateral. It is mild at first, but soon becomes sharp, lancinating, shooting and agonizing, and while at first is at intervals, it later becomes constant. This may be replaced by girdle-sensations. The root-pains of the early stages are present as well when the tumor is situated in the anterior part of the cord as when it is upon the posterior portion. It always arises from compression of the posterior roots. It will be entirely absent if the roots are initially destroyed since it is their irritation preparatory to destruction which causes the pain. The segmental pains next appear, and they are due to compression of the cord. Segmental and root-pains may be combined. They are known to be signs of compression of the segments, since they are manifested in pains in the regions of the surfaces which are known to be innervated from definite segments. If the cord is attacked initially, anesthesia is an early symptom, and yet pain of an agonizing, lightning-like character is felt in these insensitive areas. These pains may persist for days or weeks in one spot. It is *Anesthesia dolorosa*, like that in *Tabes*, and the final condition, after paralysis has ensued, is called *Paraplegia dolorosa*. Peculiarly true of paraplegia resulting from carcinomata of the vertebrae. This stage may last for months, and years even, with no further symptoms. The spinal pain is generally manifested at a point an inch or two below the site of the tumor. The nerve-trunks, with all this pain, are rarely tender.

The next symptom is an increase of the reflexes of the parts innervated from segments below the tumor with a degree of rigidity greater than is seen in any other condition. As has been said, if the tumor is situated laterally we may find a *Brown-Sequard* type of paralysis at first (sensation abolished upon one side, and motion in the like area upon the other), but it is soon replaced by a spastic paraplegia. If the compression is very severe we shall get the symptoms arising from a complete severance of the cord from any cause, i.e., lost reflexes and a flaccid paralysis. If a tumor specifically attacks an anterior root or roots before it produces any compression of the cord, we get primarily symptoms of motor irritation in the muscles to which these roots are distributed, and these symptoms are tremor, muscular spasms, contractures, and sometimes general tonic spasms with opisthotonos. Finally, in almost all cases, there is marked spastic paraplegia with contractures and deformities of the lower limbs, but unless there is the extreme compression of the cord above referred to, simu-

lating a complete division, there is never a complete motor loss, nor a complete loss of the reflexes. When this stage of definite spastic paraplegia has been reached, the parts begin to become anesthetic, and finally completely lose their appreciation of sensation, and a band of hyperesthesia is found above that of anesthesia. There is an imperative desire to urinate at first, and to evacuate the bowels, which is later replaced by an incontinence, or a paradoxical incontinence, which is a distention of the bladder with constant dribbling of the urine. If a tumor is situated in the cord itself, the parts innervated from the involved segments will be in a state of flaccid paralysis, with atrophy, and the reaction of degeneration to the electrical current. The disease may be marked by acute attacks of meningitis or myelitis, as secondary effects of the activity of the morbid growth, and in such cases the symptomatology will be modified by this fact. There may be anesthesia of the skin with analgesia, and there may be an absence, of the perception of heat and cold, or the lack of pain and temperature sensation may occur without any loss in the perception of touch. This dissociated loss of sensation is not always due to the fact that the tumor is intra-spinal, but when it occurs in extra-spinal tumors it is temporary. Herpes zoster and trophic changes are sometimes seen in the area of affected nerves. When a tumor has become fully developed it is sometimes found that the spine over its site is tender to touch, to heat, and also to the electrical current. The characteristic pains are not absent in more than ten per cent. of the cases, and are apt to be present through the whole course of the disease. This pain in the spine is more apt to be present if the tumor is seated upon the dura than when in any other location.

DIAGNOSIS. This must be considered under three heads. First: is the cause of the symptoms a tumor? If this is answered in the affirmative the next question is this: what is its nature? Thirdly: where is it situated?

DIAGNOSIS OF THE CHARACTER OF THE DISEASE. We should be led to believe that a tumor existed, if a unilateral intercostal neuralgia developed into a bilateral attack in the same locality. If later a definite girdle sensation with agonizing, constant neuralgic pain developed itself. If segmental symptoms developed in related portions of the body; and it would amount to a certainty, if at a later period a spastic paralysis developed in the parts of the body below the site of the original pain.

DIAGNOSIS OF THE VARIETY OF GROWTH. If a patient is tuberculous, cancerous, syphilitic, or has sarcoma in some other part of the body, there is a vast probability that the growth in the spine will follow the lines of the pre-existent cachexia. The rate of developement is a sign of some value. All malignant tumors are very rapid, tubercle and syphilis also, but in the two latter the

history is so illuminating that other data are relatively insignificant. Benign tumors are slow, and are also peculiar in not producing any cachexia.

DIAGNOSIS OF SITE. Tuberculous tumors are generally intra-spinal, as also are gliomata. Fibromata, psammomata or lipomata are extra-spinal. Gunumata, carcinomata, and sarcomata are either intra- or extra-spinal. The histological character of the tumor, and the question of whether it is intra- or extra-spinal is always a matter of some doubt. The question of its vertical level is more easily approximated. The situation of the pain is dependent upon the position of the tumor; it is segmental or root, and is felt in the related parts of the body, and not in the spine itself. The initial pain from a tumor in the cervical region is felt in the shoulder and neck, while if in the lower cervical region, is felt first in the hand and forearm. Dilatation and contraction of the pupils occur if the tumor is high in the cervical cord. The paralysis is also peculiar in being a hemiplegia at first, and later a quadriplegia (of all four extremities), the paralysis of the upper limbs being spastic or atrophic, or a combination of the two. In a tumor of the sixth dorsal it is felt first in the nipple and chest, while if in the tenth dorsal it is first felt in the abdomen and groin. If it is the upper part of the lumbar cord, there is an early loss of the knee-jerk. A general indication of an implication of the lumbar region is a paralysis with atrophy, and all the signs of a lumbar myelitis. Severe pains in the lumbo-sacral plexus call our attention to this region. Brown-Sequard paralysis followed by a paraplegia, or analgesia, or anesthesia, or a limited muscular atrophy of early appearance indicate that the growth is within the cord. Tumors in the cauda equina are indicated by a wide diffusion of the pains, but are most marked in the pelvic region. They involve the anus and rectum, and also the sciatic nerve, and its area of distribution. It may produce a degenerative paralysis (the lower neuron type) in the area of the ischiatic plexus, but cases have been seen when the only seat of the paralysis was the bladder.

Tumors which involve the posterior tracts may produce ataxia. Multiple tumor is indicated if there are several points which are the seat of intense pain with a mixed type of paralysis. This will result from the fact that one tumor may involve a reflex arc, in which case there will be an area of flaccid, degenerative paralysis, while another tumor may be located above an arc lower in the spine, and thus produce a spastic form of paralysis in the parts below it.

DIFFERENTIAL DIAGNOSIS. Caries of the Spine. Stiffness of the spine, great tenderness over the spinous processes, marked increase of pain on motion, or walking, general constitutional evidences of tuberculous disease, peptonuria, are present in caries

and not in tumor. When kyphosis appears we know that it is caries. Pain on rotation, percussion, or jars of the spine is also a sign. Symptoms are initially bilateral, and root-pains not so severe. Thickening of the vertebrae, and deformities and pain accurately follow the nerve-trunks.

Carcinoma of the Vertebrae. This is more difficult of diagnosis, until the disease of the vertebrae is palpable, and the rapid advent of cachexia points to it. Gowers says that growths in the bones cannot be diagnosed at first, but later the pain from movements in the spine is much more intense than in tumor, unless the tumor is in the cervical region, where its detection is comparatively easy. The pain from the compression of a nerve-root as it passes through the foramen is much greater than when the compression is within the canal. The actual diagnosis must generally rest until the external enlargement betrays disease of the bone. Starr does not agree with Gowers that the pain in carcinoma is greater than in tumor, but he thinks that in carcinoma it is more commonly felt in the spine itself, as well as in the nerve distribution, and that herpes zoster is rarely seen except in tumor.

Pachymeningitis Cervicalis Hypertrophica. This produces a condition somewhat analogous to tumor, and when diagnosis is possible it arises from the fact that tumor produces initial, and unilateral disease of the nerve-root, while cervical pachymeningitis is bilateral from the beginning, and consequently pressure symptoms upon the spinal cord are more marked in tumor than in pachymeningitis. Pains in the arms are much more severe, and there is a much earlier, and more widespread impairment of all forms of sensation in pachymeningitis.

Transverse Myelitis. Usually sudden in onset, and not especially painful, unless the meninges are affected, and even in that case the pain is not in the spinal roots, but is diffuse, and is intense over the spine; bedsores, and trophic disturbances are early features of myelitis, and late ones in tumor. Severe initial pains, with paralysis as a much later symptom, suggest tumor rather than myelitis. A very chronic transverse myelitis may produce many points of similarity.

Aneurism of the Aorta. This may press upon the vertebrae, and set up necrosis with a resulting pressure upon the cord. It becomes an intra-spinal tumor in fact. The means of differentiation are obvious, but the possibility must be kept in mind.

Sciatica may suggest a tumor of the cauda equina, or vice versa. Tumor here is bilateral from the first, while any bilateral sciatic pain must be considered to arise from some growth in the pelvis or spinal canal. The added paraplegia, and root-pains in other localities, soon clear up the diagnosis.

COURSE AND PROGNOSIS. The course of the disease is usually slow, and may take some years, although the rapidity of the

malignant conditions has been noted in the previous section, tubercle probably being the most rapid.

PROGNOSIS follows this order: Remission has sometimes been seen, for unexplained reasons, complete helplessness with extreme pain is a natural tendency, and death is usually the result of the accompanying bedsores, myelitis, cystitis, pyelonephritis, or septicemia. Operation, while always in a sense exploratory, has been fairly successful. A cervical localization of a tumor gives the worst prognosis.

TREATMENT. This is practically surgical. When the tumor can be accurately localized, and is thought to be single, laminectomy should be performed, as the damage to the cord is quite often merely secondary to the pressure of a tumor arising from the meninges or the bones. If such a tumor is found to be inoperable, laminectomy will still conduce to the comfort of the patient by relieving the spinal pressure, and by giving the tumor equal chance to extend outward rather than inward, and reduce to that extent the injurious effects of pressure upon the spinal cord.

THERAPEUTICS. As has been pointed out, the Iodides, or a full specific treatment, have proved efficacious in some cases of syphilitic tumors, and post-mortems have shown that this form of treatment may be curative in the event of glioma. It should be employed tentatively in all varieties. There is no ground for believing that we have any specific indications for any of our remedies. It is not a condition where the symptoms are capable of very successful interpretation. If symptoms obtrude themselves, which seem very characteristic of the symptomatology of a certain remedy, it should certainly be employed, and a cure is possible. The usual domain of therapeutics is entirely in the relief of pain, and this should be procured, whatever be the drugs demanded.

DISSEMINATED SCLEROSIS.

MULTIPLE SCLEROSIS—MULTIPLE CEREBRO-SPINAL SCLEROSIS— SCLEROSIS EN PLAQUE.

DEFINITION. This is a rather rare degenerative disease, which may produce scattered areas of sclerosis in all portions of the central nervous system, but most definitely involves the spinal cord and the brain. The sclerotic changes are present in a variable degree in all cases in the bulb, and the ganglia of the brain. The result is a spastic paraplegia, complicated by a peculiarly jerky incoördination in the upper parts of the body, and by bulbar and cerebral symptoms. Frequent remission of all symptoms is a characteristic of the disease. Cases are very apt to be atypical, and are difficult of recognition in the early stages.

THE SYMPTOMS OF A TYPICAL CASE. There is a spastic paralysis of the lower limbs, to which is added a tremor of the arms of the intention type, staccato speech, nystagmus, and changes in mentality.

DIFFERENTIATION from Hysteria, Primary Lateral Sclerosis, Amyotrophic Lateral Sclerosis, Ataxic Paraplegia, Hereditary Ataxia, Pseudo-Sclerosis, Tabes, Encephalitis, General Paresis, Cerebro-spinal Syphilis, Cerebral Tumor, Bulbar Palsy, Toxic Tremor, Paralysis Agitans.

SEX. There is probably no difference in the vulnerability of the sexes, but some authors claim that females are more susceptible.

AGE. Almost all cases occur between the twentieth and thirty-fifth years, and the disease is very rare after the fortieth, but a case has occurred at the sixtieth year, and quite a number of infantile and juvenile cases have been reported.

HEREDITY. Direct heredity is rarely assumed to be the cause, but there is one instance of a female and her child being simultaneous victims of the disease. The principal hereditary influence is that expressed as neuropathic taint, and the disease is prone to occur in individuals, some members of whose family, or some of whose ancestors have been insane, epileptic, choreic, or have suffered from some other form of palsy. It is not familial, but there are instances where several children in a family have been affected.

RACE. While we cannot ascribe any numerical preponderance to any given race, it is a fact that cases are more numerous among the older civilizations of Europe, than in this country.

ETIOLOGY. It has followed exposure to cold, and traumatism, general over-exertion, particularly where physical over-exertion has been associated with fear, as in the struggle to avoid some impending danger; the excessive use of certain groups of muscles, as in many trades, or in those who play wind instruments. Workers in poisonous metals sometimes develop it, showing that at times it is toxemic. A rare type is caused by malaria, and is speedily cured by quinine. It has shown its initial symptoms after such infections as influenza, typhoid, smallpox, and erysipelas. Syphilis cannot be proved to be a cause. Many cases present no evident etiological cause. All these seem to be simply exciting causes, a neuropathic constitution being a necessary predisposing cause.

PATHOLOGY. It may be almost exclusively spinal, or cerebral in site, but generally it is cerebro-spinal, and the patches of sclerosis will be found to be scattered throughout the whole extent of the cerebro-spinal axis. They are grayish pink, and harder than the surrounding tissues, and may retract slightly upon section. In size, they vary from a pin's head to that of a walnut, but whole

regions, like the pons, and medulla, may be completely atrophied. These spots begin in the neuroglia, and under the pressure of its proliferation the myelin is degenerated, and finally the axis-cylinders are disintegrated, but not universally. If this process impinges upon cell areas, the cells degenerate, lose their dendrites, and there is wasting of the emergent axon, but secondary degeneration does not take place in nerves until the axis-cylinder is affected. The spots consist of connective-tissue elements, neuroglia cells, spider cells, and a mass of fine fibres. When the process attacks nerves it is pretty uniformly interstitial, and in this case the axis-cylinders are relatively uninjured. The process is generally, and always initially, confined to the white substance, particularly the motor tracts, but it may invade the posterior columns, giving rise to lesions indistinguishable from those found in tabes, but in most cases the disease is said to be systematized, and, in others, tissues are affected from being contiguous in localization, rather than similar in function. The cell areas of the ganglia are often involved, but never primarily, but as an extension from sclerotic patches originating in the white substance of the brain. From investigation of these sclerotic patches in the formative stage, it has been discovered that an occluded artery is always to be found in the center of such areas. This would indicate that an infection was the basis of the process; that therefrom had arisen a localized thrombosis of a spinal artery, and that the sclerosis was the result of this process. Complete destruction of any area is rare, since some fibres and cells are always found in a normal condition. The greater patches may extend vertically for several segments of the cord, and a vertical extension is most common, but they may involve the cord transversely to the extent of an entire cross-section. While the distribution may be as widespread or complete as has been stated, the lesions are usually so grouped and distributed that there is little interference with sensory structures, and the motor lesions are so incomplete, from the preservation of some of the motor fibres, that the symptoms are

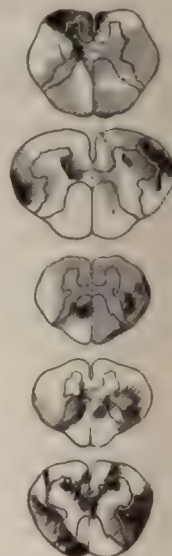


FIGURE 57.—MULTIPLE SCLEROSIS.

These sections show the variation in the location of lesions at the various levels from the lumbar to the cervical segments and in the pons.

such as would arise from imperfect conduction, rather than such complete losses as would give rise to actual paralysis. Charcot formulated a theory that the characteristic jerky incoördination was the result of the loss of the insulating myelin of the nerve fibres, perfection of which seems necessary for a perfect transmission of nervous impulses. This view was universally held, until it was pointed out that this symptom did not exist in all cases, but always, when it did exist, it was accompanied by sclerotic patches in the pons or crus. It is therefore probable that this localization is the cause of the phenomenon, and the frequency of the appearance of this symptom is due to the fact that the region of the bulb is almost always affected.



FIGURE 58. — LESIONS OF MULTIPLE SCLEROSIS IN THE CORD, MEDULLA, AND PONS.

SYMPTOMATOLOGY. If the fact before mentioned, that the disease may develop to a considerable degree in either the brain or the cord alone, is kept in mind, the atypical character of the symptom-complex will not be so confusing. The typical cases are cerebro-spinal, but a case is none the less disseminated sclerosis, if we find only symptoms dependent upon sclerosis in one only of these areas, and the others need but the passage of time to make their presence evident, since almost all cases finally become typical. Vertigo, speech defect, nystagmus, apoplectiform, and epileptiform seizures, uncalled-for laughter and tears, mental enfeeblement, and, of very rare occurrence, expansive delusions,

are all cerebral in origin. Difficulty in swallowing, paralysis of the vocal cords, atrophy of the tongue, polyuria, glycosuria, attacks of suffocation, ocular palsies and diplopia are from the bulb. A reeling ataxia, as evident with the eyes open as when they are closed, is often from a lesion in the cerebellum. Motor weakness, spasticity, the rare atrophies, disorders of the sphincters and vasomotor irregularities are from lesions in the cord. An appreciation of the point of origin of each symptom will be of material assistance in diagnosis. It is probable that motor weakness is the first symptom in almost every case, but it may be unnoted by the patient, until some striking symptom sends him to the physician, who first is called to attend a patient with an epileptiform seizure, which is followed by a transient paraplegia,

or by some generalized paralysis, which passes away in a few days. This would indicate that the brain was affected at an early stage. The most usual initial symptom is weakness of the extremities, lower most frequently, while at the same time the legs are stiff and clumsy. There is in fact some grade of spastic paraplegia. The arms, as has been said, may be the initial point of attack, but much more rarely than the legs, and, to whatever extent the case develops, the condition of the arms is usually that of spastic weakness. Diplopia, or weakness of vision, or some form of ocular palsy, sometimes marks the beginning of an attack, and one case at least, was initiated by sharp pains in the legs. To describe a typical attack, we would say

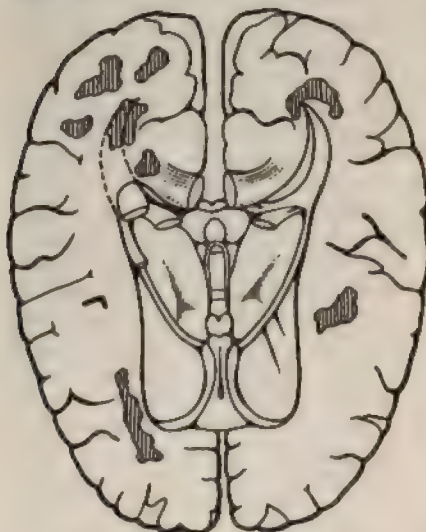


FIGURE 59.—MULTIPLE SCLEROSIS.
Lesions in the Brain.

that the patient first suffers from a spastic paraplegia; his gait is very notably disturbed from the weakness of his limbs and the stiffness associated with it; this would naturally make him clumsy in his gait; he has titubation, from the fact that the use of the muscles sets up a spasm in them; there is an ataxia of some form. The variety of this ataxia will be dependent upon some factors not hitherto referred to. If it depends entirely upon a lesion of the motor tracts it will be a clumsy stumbling gait, the legs are adducted, and crossed over one another. If the cerebellum, on the contrary, is the point of earlier invasion, the ataxia

will be cerebellar, which is a peculiarly reeling drunken form. With a coincident lesion of the motor tracts, and of the cerebellar, we shall get a combination which is called a cerebello-spastic ataxia. While not a usual site of sclerosis, the posterior columns are not immune, and, in such a case, we shall get an ataxia like that of tabes, girdle-pains, anesthetics, and sphincter disturbances, all depending upon sensory lesions. The question of implication of the motor tracts can readily be decided by the discovery of weakness and spasticity in the limbs, but between the other two we shall decide that the ataxia is cerebellar if the reeling is as conspicuous with the eyes open as when they are closed, for, if it

depends upon a lesion of the posterior columns, as in *tabes dorsalis*, gait and station are very much improved if the patient can correct his movements by the use of his eyes. After the legs have become weakened and spastic, we shall find that on attempts at movement they shake, and sometimes on being touched, which condition is called spinal epilepsy. There is an uncertainty in movement, which is most marked in the upper limbs. When fully developed this is the most conspicuous symptom, and is the well-known Intention Tremor. Passive, and intention, are the two varieties of tremor. Passive is where the part shakes with some rhythm while at rest, and the part generally grows steady on attempt at motion, and is the form typically seen in paralysis agitans. In the tremor of multiple sclerosis, on the other hand, it is not evident when the part is at rest, but on any attempt at movement it becomes noticeable, and as the object seems on the point of attainment it becomes wider, and wilder. It is also aggravated by being the object of attention, or if the patient is emotionally disturbed. It is easily investigated by directing the patient to drink from a glass of water, when he may utterly fail to grasp the glass, or, having seized it, he throws its contents about the room in attempting to carry it to his lips. In writing, the pen may be broken by the violence of the movements. Slighter, and less conspicuous incoördination of movement may also be discovered by directing the patient to extend the arms from the body, and with closed eyes to bring together the tips of the extended first fingers, at arm's length, in front of the body. The entire disability results from an interruption of the regular innervation of the muscles requisite for coördinated movement.

The possible variation in the site of the lesion is too great to allow any broad statement of the condition of the reflexes to be entirely true, but where spasticity is predominant, the deep reflexes are exaggerated, and Buzzard states that Babinski's toesign has never been absent where it was carefully searched for.

Whenever the posterior columns are affected, the superficial reflexes are abolished.

Nystagmus is an oscillation of the eyeball, usually in a lateral direction when the eye is turned to an extreme inward or outward position, but possibly only when in the extreme upward position. In some instances it is evident when the eyeball is in a position of rest. In very infrequent cases the oscillation is rotatory. The symptom is of easy determination, and is rare in any other condition, although it has been seen in acute meningitis, rarely in epilepsy, Friedreich's ataxia, cerebellar, and pontile growths, hydrocephalus, *tabes dorsalis* in an advanced stage, congenital spastic paraplegia, and congenitally in some children, without significance, or known reason. Its presence rests upon the same interference with steady innervation noticed in other parts of the body.

Speech-defect. Late Symptom. This may not be seen, but is distinctive. It is spoken of as staccato, scanning, or syllabic utterance. The voice may be hoarse. The person is deliberate and monotonous at first, pronouncing the separate syllables in an explosive manner, and becoming hurried, and finally incoherent. With it there may be tremor of the face, tongue and also of the vocal cords. There is a halt also between the words. Since it has been a very pronounced symptom, in the absence of any reasonable post-mortem explanation, it has been by some ascribed to cerebral changes.

Ophthalmoplegia. Squint is found at times and any, or all of the ocular muscles, may be the seat of a paralysis of a transient duration. This symptom is quite uncommon, 17%, and is generally bilateral, and most apparent in combined movements of the eyes, showing that the lesion is in the nuclei.

Optic Atrophy. This is common, and may be preceded by optic neuritis. It has been said to be present in 70% of all cases. From the usual localization of the lesion in the front of the chiasm, the result is almost always a bi-temporal hemianopsia with a white atrophy, instead of the gray variety, which is the usual result of implication of this nerve in spinal lesions. It is sometimes unilateral, but rarely in any form productive of total blindness, and is liable to be transient or intermitting, changing from day to day, or entirely disappearing after a time. The reduction of vision may be irregular in area, it may be a diminution of the white area alone, and there may be an irregular loss in the appreciation of different colors. When colors are lost, red and green disappear before blue and yellow, which is to some extent diagnostic, since in hysteria the reverse is the rule in color-loss. The partial character of the visual losses depends upon the fact that the site of the lesion is such that the macular bundle, the seat of the most acute vision, is almost certain to escape. The pupils may be contracted, and irregularly so; they may react poorly to accommodation, and perhaps to light, but they never actually lose the light reflex, and the Argyll-Robertson pupil (response to accommodation, but not to light), is never seen.

Vertigo. This is a common symptom, and may be an initial one; it is usual at some stage of the disease, but if early in appearance it may disappear later. It arises from defect in parallelism of the eyes, or from a lesion in the brain or bulb. If it is ocular in origin, it may be neutralized by covering one eye, but if it is cerebral this measure will not improve it.

Bulbar Symptoms. The bulb may be affected at an early stage, but usually its symptoms are of late appearance, and often are the cause of death. If the floor of the fourth ventricle is the seat of a patch of sclerosis, and it is a common site, the resulting disorders of respiration and the action of the heart prove quickly

fatal. The more general symptoms, which may be tolerated for a time, are like those of bulbar palsy. There is difficulty in swallowing and in mastication. The tongue becomes paralyzed to a variable extent, there may be wasting, and the lips and lower face tremble in speaking. Other results are polyuria, and glycosuria.

Sensory Symptoms. As a rule sensation is not disturbed, but neuralgic pains in the legs have been noted as initial symptoms at times, and neuralgic pains in the course of the disease are not rare. The sensory changes as a rule are subjective, and indicate that the brain and the spinal centers are affected, and take the form of numbness, and tingling, formication, and heat, or a full sensation in the limbs. Gastric crises, girdle- and lightning-pains are possible, but much more rare of occurrence, and indicate a lesion of the posterior columns. With a lesion in these areas the appreciation of touch, heat, cold and pain may be diminished to any degree. Headache is common, but intermittent.

Special Senses. There is often a diminution. Vision frequently fails, as has been said, and smell and taste, but deafness is quite rare.

Mental Changes. The intellect is almost always reduced in acuity, and rarely the patient is the subject of expansive delusions. There is a similarity between the physical and the mental losses. Muscular coördination requires the simultaneous action of many muscles to insure a steady and well-proportioned muscular result. In the same manner, a mental concept is the result of an extensive coördination of separate parts of the brain, and the presence of spots of sclerosis in the conduction-tracts, i.e., the white substance, which is their initial site, is fatal to correct mental efforts. The mind may be reduced to dementia. Emotionalism, shown in involuntary tears, or laughter, is common, and indicates an invasion of the basal ganglia by the sclerotic process, which we know is a common occurrence.

Among the more uncommon effects, we sometimes see slight visceral changes, and failures in nutrition. The muscles may waste to some extent, but they never show the complete reaction of degeneration. If the functions of the bladder and rectum are disturbed, it is usually slightly and transiently. Very rarely cases have been seen where the process had such a complete transverse distribution that the symptoms of a myelitis were present, and in this case there were rectal and vesical incontinence, the walls of the bladder were the seat of atrophic changes, and a succeeding ulceration, and bedsores developed.

Another symptom which may occur is contracture. The motor tracts are subject to degeneration, and in this case we shall find contractures occurring in from six to eight years. When this stage is reached we find the limbs spastic, with increased deep

reflexes, and contracted, as in congenital spastic paraplegia, in strong adduction.

COURSE. Owing to differences in symptoms between a cerebral and a spinal form of invasion, we can see the reason for the wide variation in duration of the different cases. The limit of duration is from two to twenty years, and the vast proportion of the cases last from five to fifteen years. Remission at any stage, and for a period from days to months, or even years, is a prominent characteristic of the disease. We may, with some advantage, consider it as divided into three stages. The first, of invasion, where we find spastic paralysis of the limbs, with titubation, or ataxia, or both, with vertigo, and perhaps also pains in the head and legs. Convulsions, or apoplectiform attacks may usher in the disease or occur later. In the second stage all these symptoms increase, and added to them are scanning speech, nystagmus, and intention tremor; defects of vision and ocular control are present by this time. Spinal epilepsy is liable now to occur, and late in this stage we may see contracture. The third stage is entered upon when appetite begins to fail, the patient emaciates, paralysis becomes so great, when the spasticity is also considered, that locomotion is impossible, and the upper limbs are helpless. The functions of the bladder and rectum are disturbed, and bedsores appear, and death closes the scene, either from a general inefficiency of the functions, or from some intercurrent affection which the weakened organism could not resist.

PROGNOSIS. If the brain is the seat of the disease, or the bulb is speedily implicated, the patient will probably die in two years, or less. In the purely spinal cases there is no immediate danger to life. Taken as a whole the cases tend only slightly to a fatal issue, except from deficient vitality. Remissions are the rule, and may be long-lasting. Cases have been known to recover.

DIAGNOSIS. If a case is typical, we find weakness of the lower limbs, with spasticity, and increases of the deep reflexes. The upper limbs are similarly affected, but to a less degree. There are nystagmus, and bitemporal optic atrophy. Speech is scanning, and there is intention tremor. Sensory disturbance is absent to any marked degree, there is no disorder of the sphincters, and there are no atrophic symptoms. Many cases are atypical and only by exclusion can we arrive at a diagnosis. Speech defect and nystagmus are not so often present as the text-books have indicated. However, if we find a spastic paraplegia in an adult, without the signs or history of a previous cerebral lesion to explain its presence, there is a strong probability that it is the early stage of a multiple sclerosis, especially if it is in a person below the age of thirty-five.

DIFFERENTIAL DIAGNOSIS. Hysteria. This may be antecedent to, or combined with multiple sclerosis, and the whole

symptom complex, its genesis and method of evolution, must be taken into account, or mistakes will be made. Hysteria is always the result of psychic influences upon a neurotic temperament, and it is possible to dispel all symptoms by a similar shock. Tremor, difficulties of speech, incoördination, paralyzes and contractures, may all be dispelled by a command, or a sudden appeal to the emotions. Complete paralysis of one part, with a normal condition of the corresponding limb on the other side of the body, is always hysterical. Tremor of all kinds, and of all parts, may be hysterical, but it is smaller in range, and accompanied by a tardiness of muscular effort, as if the patient wished to exert some degree of force. It is not so closely associated with voluntary effort, is more irregular and variable than in multiple sclerosis. Scanning speech is a possible symptom of hysteria, but is variable in amount in a single sentence, and, if present, lasts through all stages of the attack while in multiple sclerosis it is a late symptom. Color-loss is common in hysteria, but, when it occurs, blue and yellow are first lost, while in multiple sclerosis red and green are the first to disappear. These are the symptoms impossible in hysteria: nystagmus never occurs, nor optic atrophy, nor loss of sphincter control, nor the oscillation of the head and body which are so common in multiple sclerosis. Hysteria is not possible, if when the knee-jerk is exaggerated the plantar reflex is abolished.

Primary Lateral Sclerosis. While the gait is stiff and paretic, the spasticity is not so great as in multiple sclerosis; it has not the jerkiness of gait so characteristic of multiple sclerosis. The spasticity, and exaggeration of the deep reflexes, is more marked in lateral sclerosis, but in this latter disease sensation is not disturbed at all, and the control of the sphincters is perfect, except in the most unusual cases. Lateral sclerosis displays more atrophy. In rare cases, where the sclerotic patches are located in the dorsal region and a secondary degeneration has occurred below it, we must rely upon the symptoms in the upper part of the body to differentiate. Lateral sclerosis has no ataxia, intention tremor, nystagmus, scanning speech, nor cerebral symptoms. In infantile spastic paraplegia, or the congenital form known as Little's Disease, when it is paraplegic, the history will generally be a guide, but, if that is lacking, the presence of cross-legged progression on the one hand, and the absence of distinctive multiple-sclerosis symptoms on the other, will render a mistake improbable. When infantile or congenital spinal paralysis is quadriplegic there is a mental defect, but the other distinctive symptoms are still lacking. In no case, except in multiple sclerosis, does the condition remit.

Amyotrophic Lateral Sclerosis. It may be mistaken, but atrophy is more marked, spasticity is less, and sensory symptoms

are almost always absent; the characteristic symptoms in the upper part of the body are lacking, and there are no remissions.

Ataxic Paraplegia. While there may be a similarity in the condition of the lower limbs, the progress of ataxic paraplegia is steady, and it shows no intention tremor, scanning speech, nor disturbance of the brain.

Hereditary Ataxia. The diagnosis is very difficult if some of the family have multiple sclerosis, which is apt enough to be the fact for it to be considered. It is as unusual for multiple sclerosis to develop before the twentieth year as to have the hereditary ataxias develop after that period (Marie's may do so). There is a long history of cases in the family of hereditary ataxia. Friedreich's, in particular, develops in early adolescence; it has motor difficulties, but they are pure ataxias, never spastic, and the limbs are flaccid; the deep reflexes are lost, and there is no optic atrophy. Marie's type presents more difficulties, since it develops in late youth, has ataxia, nystagmus, tremor of the head, body, and limbs, mental failure, and optic atrophy is possible. The deep reflexes are also exaggerated, but there is never an ankle-clonus. The motor difficulty here also is ataxic, and not spastic, more of a cerebellar type, while in multiple sclerosis the ataxia is always attended by some spasticity. Apoplectiform seizures are not seen in the hereditary ataxias, nor do we find intermissions. The speech is defective, but it is not syllabic, and while there are choreiform jerking of the arms on voluntary movement, it is not an intention tremor, although similar to it.

Pseudo-Sclerosis of Westfal. This may reveal identical symptoms on examination, but there are differences on careful investigation, and the history of the evolution of the case is different. Psychic symptoms appear early, apathy even to dementia, and delirium. There is slowness in all movements, there is an absence of nystagmus, and the paradoxical contraction is present. This is the contraction of an extensor, like the *tibialis anticus*, when a flexor is put on the stretch, as when attempting to elicit ankle-clonus. It is elsewhere found only in paralysis agitans and the neuroses. There is a slow tremor, 2 to 3 per second, and it may be passive. Optic atrophy is not present. Post-mortem, no lesions have been found, so that it has been considered a neurosis. It is a very rare disease.

Tabes Dorsalis. This may be said to differ too vitally to be often the subject of mistake, but, since both have ataxia and incoordination, it is well to point out that the ataxia of multiple sclerosis is motor and spastic, while that of tabes is sensory and flaccid. The deep reflexes are exaggerated in multiple sclerosis, diminished early, and finally lost in tabes. Knee-jerk may be lost also in multiple sclerosis, but loss is transient. Permanent in tabes. Romberg is marked in tabes but, while the multiple sclerosis patient

may sway in station, the amount is not increased by closing the eyes. The tabetic is incoördinate, but on attempting a movement he does not break out into such a wild range of jerky muscular contractions. Degenerative symptoms are common in tabes, rare in multiple sclerosis. Fulgurating pains, anesthetics, and analgesias are rarely found in multiple sclerosis, and are usual in tabes. Both diseases are marked by frequent and long remissions.

Encephalitis, Meningo-Encephalitis, and Multiple Softening. There is a form of meningo-encephalitis which may end in multiple sclerosis. It is very difficult to detect any differences, but, in some cases, we can make a differentiation from the fact that the mental state is one of irritability, restlessness and excitement, instead of reduction only, that the tremor in writing is finer, and the incoördination is of a tremulous instead of a violent type. Speech is also tremulous, and there is fibrillation of the tongue. If the disease opens with apoplecticiform seizures, the prognosis must be guarded, since it may indicate encephalitis, beginning sclerosis, or paresis, but in the absence of alcoholism, syphilis, or a hypertrophied heart, such an attack with a transient paralysis is strongly suggestive of beginning sclerosis.

General Paresis. Intermittency of symptoms, and an irregularity of course suggest multiple sclerosis, while slurring speech, Argyll-Robertson pupils, with loss of knee-jerk (although it may be temporarily exaggerated), with the presence of grandiose delusions, or delirium, indicate paresis.

Cerebral Tumor. In general it may be said that both multiple sclerosis and cerebral tumor have tremor, optic neuritis, headache, exaggerated reflexes, and weakness of extremities, but they differ first in the fact that in tumor progress is steady without remissions. The head is not painful to percussion in multiple sclerosis, but may be in tumor. If optic neuritis is present in multiple sclerosis, it rarely reaches the stage of choked disc, it is generally partial, usually affects only one eye, and is often transient. In tumor, also, headache is generally constant, and severe, while it is incidental in multiple sclerosis. The vomiting, stupor, and retardation of the pulse, which are common in tumor, are not seen in multiple sclerosis. Both diseases have tremor, but that of tumor is rapid oscillation, and it is not intention in character. Cerebral tumor may give rise to an ataxia resembling that of multiple sclerosis. A tumor in the pons, or the crus, may cause a very similar jerky incoördination, but it lacks the remissions of multiple sclerosis.

Bulbar Palsy. Bulbar palsy has a speech defect, and there may be a loss of power of articulation, and of deglutition, and apoplectic seizures may occur. Cases have been seen, where there has been spasticity of the lower limbs, from irritation of the

descending motor tracts as they pass through the bulb. This spastic paraplegia is, however, unusual, and only partial at the most, while in multiple sclerosis it is a permanent feature, and an early one, except in those cases which are bulbar in primary localization. These cases differ from bulbar palsy in the fact that when the lesion is so localized the result is not the wasted tongue and trembling lips, and general atrophy and immobility of the lower face, but is tachycardia, dysarthria, instead of a slurring speech, and embarrassed respiration. In bulbar palsy, also, the laryngeal reflex is lost, not in multiple sclerosis; the mind is clear, but is disturbed in multiple sclerosis. Termination in death by fault of deglutition, bronchitis, or broncho-pneumonia, in three or four years is the rule in bulbar palsy, while in multiple sclerosis life may be prolonged for twenty years. In particular, multiple sclerosis is marked by frequent remissions and intermissions, whereas bulbar palsy is a disease of steady progression, or at least there is no remission.

Toxic Tremor. This is said never to be of the intention variety, but that is not a sufficient point of differentiation. In any disease characterized by tremor, differentiation from multiple sclerosis must be made by the course of the disease, by the presence of symptoms not found in multiple sclerosis, and by the absence of symptoms which are found in that disease. All toxic tremors have associated symptoms which are peculiar to themselves, and are diagnostic.

Paralysis Agitans. This disease has tremor, rigidity, muscular weakness, and a changed mental state, often emotional, but its tremor is very rarely intention. There is a rigidity of the whole body, which is from changes in the muscles, and not from spasticity; for a while the reflexes may be exaggerated, but generally are normal. Sensation too is disturbed toward hyperesthesia, while in multiple sclerosis it is usually toward anesthesia. The trophic symptom of "glossy skin" is common in paralysis agitans, and uncommon in multiple sclerosis. Vaso-motor disturbances are notably frequent in paralysis agitans, and rare in multiple sclerosis. The face is expressionless, without tremor, but if the face is affected in multiple sclerosis it produces a tremor. The voice in paralysis agitans is high pitched, and speech is hesitating and monotonous, not scanning, nor staccato as in multiple sclerosis. The tremor in paralysis agitans is always regular, however wide it may be, and it is very uniformly located in the upper extremities, but the character of the tremor alone is not enough to differentiate the two diseases. Paralysis agitans is a disease occurring between fifty and sixty years of age, extremely rare in early life, never before puberty.

TREATMENT. This is not always futile for relief, and ought to be always kept in progress, since certain cases cease to advance,

and our care may have aided to produce this cessation. The best measures are rest, forced feeding, massage; hydrotherapy may do good, but *hot* baths are to be avoided. Electricity is a matter of difference of opinion, but is advised by some. Quiet life without over-exertion. *Do not* put the patient to bed if it can be avoided, since it has been found that a few days' rest in bed may make a hopeless paralytic of a patient who previously had fair powers of locomotion.

Electrical Treatment. It is generally believed that this disease is not favorably influenced by electrical treatment of any kind. The disease is an interstitial overgrowth, and not cellular. Growth of this kind is not as amenable to treatment as when the lesion affects other kinds of tissue. It must however be remembered that this is a disease which is not in itself fatal to life, and despondency, and resultant resistance to the ordinary diseases of mankind, is very much lessened if the patient feels that an effort is being made to remedy his condition. On that account therefore, I feel that he should have treatment of such a tangible kind, and one which meets so fully the desires of his friends. The most reasonable plan has been stated by King. He says that the current should be given in the direction of conduction of the tracts, and that therefore when the posterior columns are the seat of the trouble that the current should be an ascending one, and if the antero-lateral, it should be descending. If, however, the disease be most marked in the anterior horns, the current should be made to pass transversely through the diseased segments. He says also that the greatest effect will be in strengthening the muscles by giving them active exercise with the interrupted galvanic current, application being made over the motor points, or still better than the interrupted, is the use of the sinusoidal. Galvanism mitigates the tremor but slightly, if at all. If muscular contraction can be produced by static sparks the effect is similar, but slighter than from the galvanic. Theoretically the tremor should be helped by giving stable anodal applications over the diseased nerve-trunks.

THERAPEUTICS. No therapeutic measures have proven very beneficial, but from the fact that some cases recover under any treatment, we may believe that the number might be increased by proper treatment, and symptomatology furnishes the most rational basis. The remedies which have seemed most apt are the following, and I have usually employed them in the 6th potency. Aurum mur. when the posterior columns are also affected, shown by the presence of sensory symptoms. Argentum nit. for trembling with paralytic weakness. Baryta carb. and mur. both have paralytic weakness with tremor. Belladonna as above with diplopia and dimness of vision. Calcarea for constitutional degeneration. Causticum, paralysis with intact sensibility. Conium, weak, trembling gait with reeling (implication of the direct cere-

bellar tracts, or cerebellum). *Crotalus*, paralysis with no sensory symptoms. *Cuprum*, paralysis with spasticity. *Lathyrus*, paralysis with ataxia, and rarely a disturbance of sensibility. *Lycopodium*, paralysis with formication. *Mercurius*, general tendency to neuroglia overgrowth. *Natrum mur.*, paralysis with spasticity. *Nux vomica*, spastic paraplegia. Oxalic acid, paralysis with pains and bulbar symptoms. *Phosphorus*, paralytic weakness and ataxia. *Physostigma*, paralysis with ataxia and muscular cramps. Picric acid, weakness of the limbs, with spasms and priapism. *Plumbum*, tremors and paralysis with cramps in muscles. *Rhododendron*, paralytic weakness with formication. *Silicea*, when the posterior columns are affected. *Tarantula*, with spasms in muscles. *Thuja*, paralysis with tremor.

CHAPTER VI

DISEASES AFFECTING THE SPINAL GRAY-MATTER

ANTERIOR POLIOMYELITIS ACUTA.

INFANTILE SPINAL PARALYSIS—ACUTE ATROPHIC PARALYSIS.

DEFINITION. It is an acute form of paralysis, characterized by an immediate general paralysis, or of one or more limbs, which is soon replaced by a selective paralysis with rapidly succeeding atrophy. Persistent sensory symptoms are rare, but, if present, they consist of pains confined to the back and the affected muscles, and are of slight intensity, and transient in duration.

THE SYMPTOMS OF A TYPICAL CASE. After a slight and transient fever, a young child is found to be generally paralyzed in the upper or lower limbs. Pain is slight, or absent, and there is no disturbance of the mind, except in a possible convulsion at the outset. After two or three weeks, the paralysis becomes strictly limited to a part, or a few muscles of a part, and these muscles will be found to be atrophied.

DIFFERENTIAL DIAGNOSIS. To be diagnosed from Palsies of Cerebral origin, Transverse Myelitis, Pseudo-hypertrophic Paralysis, Progressive Muscular Atrophy, Hemorrhage into the Anterior Horn, Cerebro-spinal Meningitis, Multiple Neuritis, Rheumatism, Rhachitis, Congenital Dislocation of the Hip, Hip Disease, Erb's Palsy.

AGE. Rare before the fifth month. Less so between the sixth and eighth, and almost all cases occur between the second and third year of life. It has however been seen as early as the twelfth day of life, and sometimes occurs in adults. One of the author's cases was a female thirty-five years of age. Congenital clubfoot has been ascribed to its occurrence *in utero*, and in such cases there has been found a deficient development of the sacral cord.

ETIOLOGY. Cold and Trauma have been held to be causes. Recently it is more and more considered to be an infection, or secondary to infectious diseases; after measles, scarlatina, and pertussis. In all countries it is most common in the seasons of the greatest heat. It is sometimes epidemic, and certain localities are especially apt to furnish examples of it.

PATHOLOGY. To understand the symptomatology of this disease, several facts must be appreciated: (1st) separate muscles are represented by groups of cells, nutritive as well as motor, in the cord, while the grouping in a similar area in the brain represents a movement combining the cells of innervation of as many muscles as are concerned in such a movement. (2d) Such cell clusters in the cord are most numerous in the cervical and lumbar enlargements. (3d) Some groups are very long vertically, and the complete innervation of a particular muscle may extend through three or four segments. (4th) The arterial twigs from the anterior spinal artery do not divide into branches, which by subdivision go to both

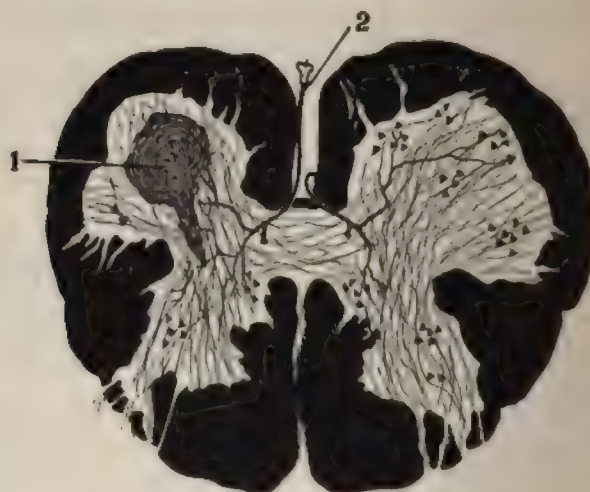


FIGURE 60.—ANTERIOR POLIOMYELITIS ACUTA.

1. Indicates the area of softening (observe the blotting-out of motor cell-groups). 2. Indicates an anterior spinal artery (observe that one artery goes to one side and another to the opposite).

anterior horns of some definite segment, but one anterior branch goes to one side, leaving the blood supply of the opposite half to come from the next branch. The plugging of one primary branch would therefore result in a degeneration in one anterior horn only, and the resulting paralysis would be upon one side of the body only, and such is the usual clinical fact. Through infection an embolism or thrombosis is produced in the anterior spinal artery, or a branch of it, and as a result exudation and hemorrhage take place, with extrusion of leucocytes and consequent degeneration of an area, generally confined to one segment, but perhaps extending through several. The cells in the horn degenerate, and their fibres waste, and the antero-lateral tracts may also degenerate.

This latter loss would explain a lack of pain in severe cases, and also the fact that growth in the affected limb is either totally arrested, or does not progress at a normal rate, resulting in deformity, or at least in a lack of volume.

SYMPTOMATOLOGY. Onset abrupt. There may be a chill, but it is often unnoticed; fever, possibly high, 102° to 104° ; vomiting, convulsions, anorexia, stupor or delirium. The duration of the fever is from a few hours to some days, or so slight as to be unnoticed, and its intensity or duration gives no indication as to the extent or severity of the subsequent paralysis. For from one to seven days the case may have only the general symptoms of an acute infection. When it subsides the paralysis may first become evident.

The paralysis which now comes on is diagnostic in form; it is completely developed from the beginning. Site: one leg most frequently, one arm next; both legs next (rare), four extremities next (but this is very rare); face, tongue and eyes are rarely if ever attacked. During this stage children are apt to cry from pain in the muscles, or from root-pains in the back and neck. The paralysis is always flaccid, always with atrophy and fibrillation to some degree. Degeneration, shown by reaction of degeneration, begins in two weeks, but mild cases show a reduction to faradism only. The state of general paralysis lasts only from one to three weeks, and at the expiration of that time we find that, instead of a limb being helpless, certain muscles or groups of muscles only in that limb will fail to respond to the will. The paralysis is remarkable in being selective in character. Lasting disorders of sensibility are slight, and usually absent. If pains persist in the muscles they are due to a secondary neuritis, or to a resulting myositis. Knee-jerk is absent if the quadriceps-extensor is affected. Sphincters are never affected except with a possible retention of urine during the first few days. Atrophy and paralytic contractures are rather common, also scoliosis, lordosis, and loose joints. Contractures are from unopposed antagonists. Vaso-motor changes occur, which render the skin cold and blue, so that the temperature will be below that of the unaffected limb.

PROGNOSIS. Good as to life. Muscles which do not lose faradic excitability after two to three weeks will recover. Muscles which show reaction of degeneration after the first week will not recover, but may be improved in function.

DIAGNOSIS. Since we know that the motor cells in the anterior horns are also trophic, any injury to these horns will be displayed in a flaccid paralysis accompanied by wasting in the parts innervated from this level. When, therefore, we have a spastic paralysis with weakness and increased reflexes, we know that the lateral columns are also affected. If there is more wasting than can be

accounted for by disuse, we know that the anterior horns are also affected to some extent, and that the disease in question is developing from a pure spastic paraplegia into an amyotrophic lateral sclerosis. If the spastic element is wanting, we know from that that the lateral columns are not affected, and if it is very slow in its development, we know that it is not an inflammatory process of the anterior horns, but the wasting presupposes that they are affected, hence, knowing that it is not in the lateral columns, and that it does affect the anterior horns, and is yet without inflammation, we are forced by exclusion to the diagnosis of some form of progressive muscular atrophy, viz., poliomyelitis chronica. If this flaccid wasting palsy comes on with great suddenness, we know that we are in the presence of the results of some acute inflammatory process affecting the anterior horns alone. The only process of this character is poliomyelitis anterior acuta.

DIFFERENTIAL DIAGNOSIS. It is a most satisfactory plan in differentiating this form of paralysis from those possessing some degree of similarity to it, to divide them into those which are respectively cerebral, spinal, and peripheral in origin.

Cerebral palsies are strictly hemiplegic in distribution, tongue, face, and speech often suffer; the advent is abrupt, and there is rigidity of the limbs, the tendon reactions are increased, the electrical reactions are preserved, or only diminished in quantity, and there is no loss of faradic irritability. The convulsions, if present, are focal or affecting only one side of the body, in any event commencing locally, even if they afterwards become general. There is no extreme wasting of the muscles. In older children there may be aphasia. There is mental depression of a profound character, and there may be final deterioration. As a late result there is apt to be athetosis or hemiplegic contracture. In anterior poliomyelitis, on the contrary, the distribution of the paralysis is not generally hemiplegic, the tongue, face, and speech are unaffected, the tendon reactions disappear so far only as they are in the area of the muscles involved. There is the electrical reaction of degeneration, and faradic irritability soon disappears. There is rapid wasting of the muscles. A late result is deformity resulting from partial paralyzes of the limbs. Mentality is unaffected. If convulsions occur they are general in character.

Atrophic Forms of Paralysis from Other Causes. If the child has a transverse myelitis there will be a paralysis of sensation, as well as of motion; of the sphincters also; bedsores will occur; there will be no rapid diminution of the area of the paralysis, and there will be a long-continued fever. The electrical reactions will not be so dissimilar as to have any diagnostic value.

Pseudo-hypertrophic Paralysis. Occasionally will be confused on account of the fact that there may be a deposit of fat in atrophied muscles in late stages of anterior poliomyelitis. This

increase in muscular size is, however, a late symptom in anterior poliomyelitis, and an early symptom in pseudo-hypertrophic paralysis.

Progressive Muscular Atrophy. Rare in children. The slowness of the disease ought to differentiate it, and if not, the carved-out appearance of the atrophied muscles, bounded by well-developed ones, differs from the more general paralyzed condition of anterior poliomyelitis.

Hemorrhage into the Anterior Horn. This is a rare accident, but the results are identical, and it cannot be differentiated, except possibly by the history.

Cerebro-spinal Meningitis. The history of the attack is dissimilar. If there are convulsions they may be general, but the paralysis may be of the cerebral type, that is spastic instead of flaccid; the spinal and nuchal rigidity is peculiar to it, and an extensive degree of paralysis will not occur without a meningitis, which will give rise to far more pain than is found in anterior poliomyelitis.

In general it may be said that all diseases of the spinal cord producing paralysis, but not affecting the anterior horns of gray matter, are to be distinguished by the absence of reaction of degeneration and of atrophy and fibrillation; the paralysis is spastic as are the contractures; there is an increase in the reflexes, and mentality is liable to be decreased.

Multiple Neuritis. This is peculiarly peripheral. It is symmetrical in affecting both sides of the body; it has marked sensory disturbances, in that, aside from spontaneous pain and changes in sensation in the extremities, the nerve-trunks are swollen and tender, and there is a history of intoxication or infection, and wrist drop and foot drop are diagnostic if present. There are, however, conditions favorable to error, since the patient may be too young to give us any criterion of his sensations. It is also true that polyneuritis may complicate anterior poliomyelitis, but it may be said that toxic neuritis is rare in children, acute anterior poliomyelitis extremely rare in adults.

Rheumatism. Since both diseases commence with a moderate degree of fever, and often result in a speedy immobility of the limbs, and patients may be so young as to render doubtful the cause of this immobility, errors are sometimes made. Careful examination will disclose that the child *can* move the limb, but is *unwilling* to do so, if the cause be rheumatism. It will also be found that the limb is hot, in place of the coldness of the spinal paralysis; that the joint is tender to pressure; that instead of a blue coldness of the skin there is sweating, but no atrophy is present, which is to be expected in an early stage of anterior poliomyelitis. After some inflammatory conditions of joints secondary atrophy does take place, but the patients are generally adults, and the

symptoms localizing the trouble in the joints, and not in the spinal cord, are usually convincing.

Congenital Dislocation of the Hip. May be taken for a late case of anterior poliomyelitis, but careful examination of the hip joints will show the anatomical faults, the gait is peculiarly diagnostic, and there are none of the positive symptoms of anterior poliomyelitis.

Hip Disease. In the first two days of anterior poliomyelitis we may have pain in the back, the leg may be flexed and rotated outwardly, and it may be immobile. This would remind one of hip disease, but an examination will show that the articulation is perfect, and that the leg is capable of passive, but not of voluntary motion.

Erb's Palsy. This may appear like a case of anterior poliomyelitis affecting the cervical enlargement, but the patient will be a newborn infant, and there will be a history of traumatism during labor. Examination will show that it is a localized neuritis of the brachial plexus. There will be a localized anesthesia in the distribution of the circumflex nerve, that is, over the lower deltoid region, and the deltoid, biceps, coraco-brachialis, and supinator longus are paralyzed.

Rhachitis and Marasmus. The two conditions are similar in an unwillingness to walk, sudden onset of fever, and pain and tenderness in the limbs in children. There is no history of a preceding illness. They may complain of a certain degree of pain, or they may lack those symptoms entirely. If the cases are carefully examined it will be found that cases of anterior poliomyelitis have no tenderness of joints, though perhaps some from muscular spasm, and that the legs are paralyzed. In the case of rhachitis we shall find that there is no paralysis, but that the child is unable to walk on account of the softness and the tenderness of the bones, especially about the articulations. The pain and tenderness in rhachitis is not limited to a few muscles, but is in the bones as has been said, and generally through the muscles. The gums are tender and swollen, and there is sweating of the body. Such a child may have been brought up in the best of surroundings, but investigation will reveal a defect in the nutrition. There is another group which is marasmatic, and the difficulty in locomotion is, in this case, due to a weakness of the muscles.

TREATMENT, adjuvant. The treatment varies with the stage of the disease. In the initial stage with fever it is treated as an acute infection. The patient is to be sponged with cold water if the temperature is over 100 degrees, and this regimen is to be persisted in so long as the fever is present. Locally the indications are to relieve the congested area of the spine so far as possible. This is to be accomplished in the first place by keeping the patient on the side rather than on the back, and a board rest against which the

body may be leaned is useful. Some advise that the patient should be in a partially upright position on one of the numerous patterns of bedrests. The patient may be kept on the face for part of the time with advantage. Keeping the bowels open, and the kidneys and skin active also favors reduction of the spinal congestion. Local measures should not be used according to theoretical standards, if thereby the patient is made uncomfortable. Cold local applications are not advisable, and while heat is of value it must be continuously applied, and if the nursing is not skillful, and if there is an alternation of hot, rather warm, and then cold applications, they are of positive damage. In place of heat, mild sinapisms may be used, and kept on long enough to just redden the skin, and then removed to be replaced every three hours. One part of mustard and three parts of flour make a good formula. In place of the mustard the back may be kept irritated with iodine. Every day the skin should be gently massaged with the hand, or salt-rubs are valuable to keep up the nutrition of the muscles and the skin. From the first, the affected limbs should be wrapped in cotton, and then supported by a gauze bandage. When a limb or extremity shows a tendency to assume an abnormal position it should be restored to the normal, and retained there by sandbags, strips of plaster or by splints, remembering in the last instance that the tissues do not stand pressure without damage, unless the parts are frequently relieved from it, and their nutrition restored by massage. Massage should always be in an upward direction. Guard against bronchial inflammation for the time, since the weakened trunk-muscles do not affect a perfect respiration. No matter how light the attack, the patient should be kept warm and at rest in bed for from two weeks to a month.

When the more chronic stage has supervened the main indication is to prevent contractures, and to preserve the nutrition of muscles which will recover, wholly or in part. This is done by antagonizing contractures in the knee, for instance, by wearing a slipper at night with an elastic band attached to the toe and running to the knee. Similar attachments from the elbow to the back of the hand may be of use. Gymnastic exercises will be useful, and massage and Swedish movements. Salt-rubs are helpful, and children may be urged to play in baths at 90 to 95 degrees for an hour or two each day. Simple, energetic massage by some one in the home has often worked wonders. The persistency and regularity of massage and simple exercises are the main hope, and they may be done as well at home as by the professional rubber.

Electrical treatment. Electricity is of great benefit, but it does not delay the wasting or prevent it, nor can we directly affect the cord, but it does keep up the circulation in the muscles, and preserves the nervous mechanism in an approximation to functional activity. It may be said to be of universal applicability, for

in cases of long standing, even of many years, it will stimulate cells which still retain some degree of vitality, and will endue with new power muscular fibres which have been useless from neglect. It will not rejuvenate cells which have been destroyed, or muscular fibres whose nerve supply has been destroyed. The electrical current may be used for diagnostic purposes at any time, yet caution should be exercised, but the muscles should not be treated therapeutically so long as they are tender, and the spine should not be treated until the temperature has been normal for eight days. The muscles which are capable of restoration will first show it by a slight swelling-up as the current passes, and a further improvement is shown by a response to the faradic current. Treatment should be first addressed to the spinal segments, and a positive electrode large enough to cover the involved segments, and the spinal roots therefrom, should be placed over the spine, and the negative upon the abdomen. Pass a current up to five milliamperes for five minutes, and gradually reduce it to zero. Give a treatment every other day for two weeks, and then treat with a reverse of the polarity, and an increase in the current to ten or twelve milliamperes. Still later this same application will be found more stimulating if an alternating current is used. After the disease has become chronic, the muscles should be treated with the galvanic current every other day, oftener in the later stages, and when voluntary movement is possible it should be encouraged. If the nerve is somewhat degenerated, the positive pole indicates this by its stronger response, and should be the one employed, but should be changed for the negative when this is found to produce stronger contractions. The interrupted galvanic is the form to be employed, and at first the interruptions should be very slow, as the muscle is very readily fatigued. In all cases the current should be so slight as not to frighten the patient, and nothing more than electrical massage may be possible for some time. At first, whatever be the rate of interruption, only a few contractions should be demanded of any muscle. When the muscles will respond to faradism, home treatment becomes possible, and may be given for from five to ten minutes twice a day. The interruptions should be slow, and the current just strong enough to produce contractions. While the galvanic current is theoretically better, the possibility of sufficient treatment may be beyond the financial resources of the parents. The more frequent faradic treatment may then be the preferable one, on account of the relatively low cost, and simplicity of a faradic battery. Each electrical treatment should be terminated by a vigorous upward rubbing of the affected limb with the hand, previously lubricated with some form of animal fat.

THERAPEUTICS. In the first stage if the patient has fever, especially if there is tingling of the extremities, Aconite. Dis-

continue with the first appearance of paralysis. If the onset is sudden, the fever high, and continues after the advent of paralysis, Belladonna. If it succeeds an infection, and the patient has a feeble pulse, reduced temperature, and a tendency to ptosis, Gelsemium. If in a post-infection case we have symptoms of great restlessness, and with it muscular soreness, we should think of Rhus tox, or Arsenicum, sometimes Lachesis. With a hard, bounding pulse, or, on the contrary a feeble but rapid one with much cerebral excitement, Veratrum vir.

After the initial fever the best indicated remedy is Plumbum. The 30th is strongly advised. It is indicated in all but the earliest stages. Where the spinal symptoms are persistent, and spasms are a feature of the case, Cuprum. In an intermediate stage Phosphorus may be of value, on account of its aptness to any atrophic condition. Mercurius may be indicated, since its action is on interstitial tissues, and this is a disease where there was a primary inflammation from vascular occlusion. Secale has been advised, and in this case has been found to be of greatest value when used in tangible doses, viz., ten drops every four hours for a child of two years and below, and an increase of two drops to the dose for every additional year of age. In late cases Baryta carb., and Causticum and Graphites have been advised, the latter on account of its action on scar tissue.

PROGRESSIVE MUSCULAR ATROPHIES AND DYSTROPHIES.

DEFINITION. These are both diseases which, with few exceptions, are characterized by an atrophy, which precedes and is the cause of a paralysis of a variable degree and extent. The atrophies differ from the dystrophies in the locality of the original degeneration. Every muscular atrophy depends upon some degeneration of the nerve-cell, and is therefore termed a Myelopathy, while a dystrophy arises from a primary pathological process in the muscular fibre itself (instead of that being a secondary result of some initial process in other structures), and is therefore called a Myopathy.

Every muscular atrophy is due to a disease of the lower or peripheral neuron, and may further be either primary, or secondary. If primary, it is an original disease of the cells of the anterior horns, and is what Charcot termed the Protopathic type, and is the pure type of muscular atrophy. The secondary form, called by Charcot the Deuteropathic type, has also a wasting of the cells of the anterior horns, but it is dependent upon a preliminary disease of the pyramidal tracts. This secondary form

is also known as Amyotrophic Lateral Sclerosis, and is described under that head.

PROGRESSIVE MUSCULAR ATROPHY. THE SYMPTOMS OF A TYPICAL CASE. There is the gradual wasting of a part of a limb, or several parts of the body, producing a paralysis, without sensory changes, and without an initial fever, or any general disease to explain its presence. A Progressive Muscular Dystrophy differs only in its earlier phenomena. There is a pseudo-hypertrophy in the muscles with a progressive weakness before the atrophy, which finally becomes as pronounced as if the atrophy were the initial symptom.

AGE, SEX, AND HEREDITY differ so widely in their influence upon the development of the different forms that they will be referred to as the separate divisions of this disease are described.

ETIOLOGY. No cause can certainly be proven, but it is probable that a fault in embryonic development lies at the root of many, and perhaps of all cases. On this as a basis, exposure to cold, deprivation from food, the intoxications and infections (syphilis probably not efficient), and emotional shocks or physical injuries are any one of them sufficient to produce the condition. In some cases the degeneration alone is evident, while occasionally, an extensive endarteritis is the basic pathological change.

PATHOLOGY. Progressive Muscular Atrophy is caused by a simple shrinkage of the lower motor neuron. That includes cells, dendrites, and axons alike. There is no change, such as we find in the ordinary degenerations, but, preserving its usual form, the axon becomes too small for its lymphatic envelope, and finally, there may be only a rounded, nucleus-like body, or small mass of granules, to mark the former site of a cell. The dendrites, which formerly were collectors of nervous impulses from nerve-terminals in the neighborhood, and axons, which transmitted motor and trophic impulses to the structures to which they were distributed, also shrivel and become inert. This applies not only to the large and small motor-cells, but also to those which give rise to the association-tracts. The cells of the column of Clarke are also shriveled, and so the whole antero-lateral part of the cord is shrunk. In the muscles, there is a simple atrophy of the fibres, with a loss of the striation, and fatty deposits are also found. Occasionally, an hypertrophied fibre will be discovered.

MUSCULAR DYSTROPHY, on the other hand, arises from an initial change in the structure of the muscles themselves, which goes on until the muscle is degenerated. This is, in almost all cases, preceded by an hypertrophy. This may originate as an actual enlargement of the muscular fibres, or an increase in the bulk of the interstitial tissues, or it may be, and usually is, some combination of the two conditions. Whether hypertrophy has preceded atrophy or not, whether the muscle-fibre, or interstitial

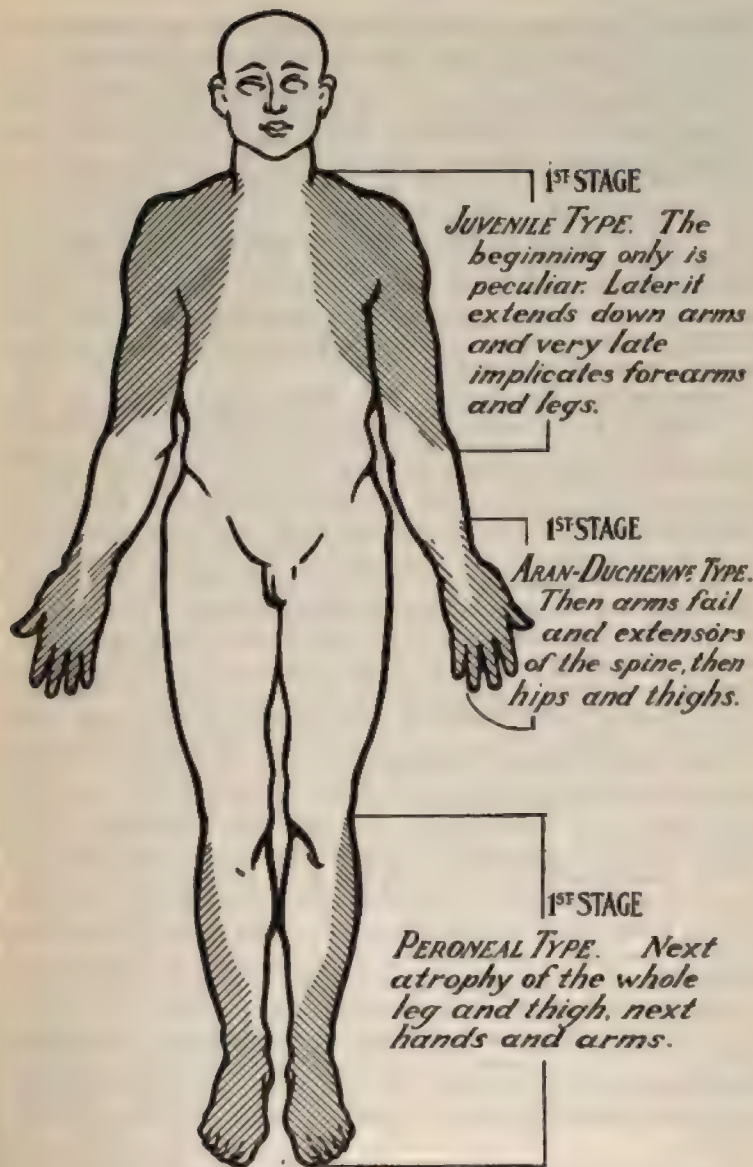


FIGURE 61.—MUSCULAR ATROPHIES.
(Modified from Mettler).

tissue has been the site of the initial change, the final result is the same. The muscular tissue loses its distinctive appearance and quality, and may be found in any stage of degeneration, even to being replaced by drops of fat. Some fibres will shrink from their ordinary size of 5 centimetres in length, and 5 to 15 millimeters in breadth, to mere threads, while others, as has been said, will altogether disappear. In the narrowed remnants, some striation may remain, but they will be farther apart than is normal, as if the weak muscular thread had been stretched in its effort to functionate. It appears to be a developemental fault, since in embryonic life there is an excess of fibres, which are probably normally removed in somewhat the fashion above described. In this disease, a normal process has been carried out to an excessive degree at too late a period.

DIFFERENTIAL DIAGNOSIS. This pathological statement does not comprise all the evident differences between the two conditions. Clinically, we find that a Dystrophy differs in these points from an Atrophy: (1st) Age at onset is earlier. (2d) Hereditary influence is generally traceable. If present at all in an Atrophy, it is generally through the paternal line, while in a Dystrophy, where it is usual, it comes through the maternal line. (3d) The point of initial attack is different, since in a Dystrophy the hands do not suffer first, but, on the contrary, the large muscles of the arm, the shoulder, or of the pelvic girdle. (4th) Bulbar paralysis does not occur. (5th) Muscles do not waste in groups, conforming to the localization of their nervous supply in the segments of the cord. (6th) Apparent hypertrophy of some muscles may co-exist with atrophy of others, or there may be a congenital absence of some muscles. (7th) Fibrillary twitchings are, as a rule, absent. (8th) There is never a typical reaction of degeneration, but, as muscles waste, there is a diminution in electrical response to an equal extent to both the galvanic and faradic currents. (9th) The deep reflexes are not exaggerated, as in the deuteropathic form (Amyotrophic Lateral Sclerosis), but if the quadriceps extensor is wasted, the knee-jerk will of course disappear. These pathological and clinical differences very definitely separate the atrophies from the dystrophies, but there are in both grand divisions symptom-complexes which are quite definite, and so differ among themselves in method and site of onset, rate, and law of extension, and effect upon future activity, and even upon life itself, that some sort of subdivision has been forced upon every writer. The following scheme has seemed to me to be the most reasonable and illuminating:

Divide the Progressive Muscular Atrophies thus:

Chronic or Subacute Anterior Poliomyelitis.

Atrophy of the Aran-Duchenne type.

Atrophy of the Charcot-Marie-Tooth type.
Hereditary type of Werdnig and Hoffman.

Divide the Dystrophies thus:

Hypertrophic or Pseudo-hypertrophic type.
Erb's Juvenile type.
Facio-scapulo-humeral, or the type of Landouzy-Dejerine.
Hereditary form.

While describing the atrophies and dystrophies, it seems well to consider a class of diseases where, as yet, we can discover no tangible changes in either nerve or muscle, but in which clinical observation would lead us to believe that there had been some sort of change in the muscle-elements. These diseases are Myasthenia gravis or Asthenic Bulbar Palsy, and Thomsen's Disease.

DIAGNOSTIC FEATURES OF THE VARIOUS TYPES OF ATROPHIES AND DYSTROPHIES

AGE: INFANCY. Facio-scapulo-humeral. 2 to 4, or at any rate in early childhood.

Hereditary form of Atrophy. Before 2d year. (6 to 12 months. Ormerod.)

Pseudo-hypertrophic Paralysis. 2d to 20th year.

CHILDHOOD. Erb's Juvenile type of Dystrophy. 12 to 16 or 20.

Hereditary form of Dystrophy. 8 to 10th year.

EARLY LIFE. Charcot-Marie-Tooth type. Before 20.

ADULT. Aran-Duchenne type of Atrophy. Rarely before 20; generally 25 to 45.

LATE ADULT. Chronic or Subacute Anterior Poliomyelitis.

LOCALIZATION. (This indicates the site of initial and usual lesions.)

Facio-scapulo-humeral type of Dystrophy. Face, shoulder, arm. Later it may involve back and legs, and rarely, all parts.

Charcot-Marie-Tooth type. Calf, foot, leg, and later the arms.

Aran-Duchenne. Hand, arm, shoulder, back, (lower limbs, late, and very rarely, if ever).

Chronic or Subacute Anterior Poliomyelitis. All four extremities (neck and back late, if ever).

Hereditary form of Dystrophy. Back and lower extremities.

Hereditary form of Atrophy. Back and thighs, arms, legs, and hands.

Pseudo-muscular Hypertrophy. Calves, legs, back; later shoulders, arms (hands always free).

Erb's Juvenile type of Dystrophy. Shoulder, arm, back, leg, calf (hands free).

FIBRILLATION. All Atrophies.

REACTION OF DEGENERATION. All Atrophies.

HYPERTROPHY OR PSEUDO-HYPERTROPHY. Pseudo-hypertrophic paralysis. Erb's Dystrophy.

HEREDITARY. All Dystrophies and Charcot-Marie-Tooth type of neural Atrophy. Dystrophies through the maternal line; when an Atrophy is hereditary it descends through the Paternal line.

CURABLE. Chronic and Subacute Anterior Poliomyelitis, sometimes.

FATAL. Chronic and Subacute Anterior Poliomyelitis, sometimes. Hereditary type of Atrophy before or by the 6th year.

COURSE. Rapid. Chronic or Subacute Anterior Poliomyelitis. Hereditary type of Atrophy does not last over 4 years. Slow. All other Atrophies and Dystrophies.

CHRONIC OR SUBACUTE ANTERIOR POLIOMYELITIS.

(Atrophy of the Arm and Leg Type.)

DEFINITION. An atrophy of a late and rare form affecting the arm or leg, and finally all the extremities.

THE SYMPTOMS OF A TYPICAL CASE are these: A person in late adult life develops an atrophy in a muscle or in a member, which spreads over all four extremities. This process spares the trunk, diminishes the deep reflexes, displays the reaction of degeneration, but does not disturb the sphincters or sensation, and while it may be fatal to life is sometimes curable.

AGE. Late adult life.

SEX. Sexes about equally. Not familial.

ETIOLOGY. Doubtful, but probably some form of toxemia. Has developed in diabetes; Erb says from traumatism; Gowers says intemperance, venereal excesses, cold and falls on the back.

SYMPTOMATOLOGY. While a whole limb may be attacked, it may seize upon single muscles here and there, since muscles tend to become involved as different contiguous cell-groups become, in turn, the seat of the peculiar degeneration. The atrophy tends finally to involve the four extremities, and, in some cases, the trunk and the bowels are also the seat of atrophy. The deep reflexes are diminished, but the sphincters are spared, and there is no diminution of sexual power. The atrophy is preceded by fibrillation. Electrical changes may precede the atrophy, and while the complete reaction of degeneration is to be expected, the

preservation of some fibres of a muscle may render it only partial in some localities. The only sensory changes are occasional pains simulating rheumatism.

COURSE. Invasion period is from two weeks to some months, and then the disease may halt, or show a gradual extension.

PROGNOSIS. The slower the invasion the slower the subsequent progress. Cases arising from traumatism are most likely to reach their full developement in six or eight months, and then may rest at that stage of debility, or slowly improve to some extent. Debility may be stayed at any point, or the person go on to complete helplessness, but with no tendency to death, or may die from pneumonia, or cardiac debility.

ARAN-DUCHENNE TYPE.

(Shoulder-arm-body type.)

DEFINITION. This is the usual form of a muscular atrophy, picking out the muscles in a selective fashion, and producing a very extreme grade of disability. It is very apt to invade other parts of the cord besides the cells of the anterior horn, and adds some symptoms of the involvement of the upper neuron. Gowers says, that he has never seen it pure in type.

THE SYMPTOMS OF A TYPICAL CASE. A man in the prime of life develops very slowly an atrophy in the muscles of the shoulder, then the arm, and possibly of the body, which spares the legs. There is some degree of reaction of degeneration. It is incurable; it may remit, and may be fatal.

AGE. Generally from twenty-five to forty-five.

SEX. More males.

HEREDITY. Not a factor.

ETIOLOGY. Not always known, but may follow traumatism (often a blow on the neck), infections, intoxications, and exposure, and sometimes complicates tabes dorsalis.

PATHOLOGY. This is peculiar only in its localization. The cell-shrinkage is, at first, limited to the 1st dorsal, 8th and 7th cervical. These segments furnish the nerve-supply to the hands. After these have become severely affected, it next attacks the 5th cervical, which supplies the deltoid, and then the adjacent cell-groups, which go to the biceps, coraco-brachialis, and supinator longus. It finally involves all the cell-groups of the cervical enlargement, and then attacks the groups in the lumbar enlargement. It may become general.

SYMPTOMATOLOGY. Some rheumatoid pains may usher in the disease, and there may be a feeling of superficial numbness, or the very first symptom may be a wasted appearance of the ball of the thumb (thenar eminence), or of the thickened muscular mass

below and to the outside of the little finger (hypothénar eminence). This allows the thumb to drop back to the plane of the fingers, and the result is "Ape Hand," and as the lumbricales, and the interossei become wasted, and the articulations relaxed, the distal extremity of the first phalanges is retracted, and the result is the "Claw," or "Preacher's" Hand. Next, the deltoid is wasted, and the arm drops at the shoulder. The other muscles of the shoulder-group soon follow, and the shoulder-blades swing down and out, giving rise to the very characteristic "Angel-wing" deformity. From the atrophy of the biceps, and coraco-brachialis, the power of flexing the forearm upon the arm is lost, and the member swings loosely, often partially dislocated. The point of the shoulder is allowed to drop from atrophy of the trapezius, and serratus magnus. The wasting of the Levator anguli scapulae, inserted into the posterior border of the scapula, allows it to drop, and the lower angle to stand out—"Angel-wing." The same process next affects the muscles of the trunk and back. The legs are usually spared, but are finally as helpless as the arms, if the disease manifests its fullest possibilities. In all forms of progressive muscular atrophy, it is to be understood, that paralysis follows and is proportionate to the atrophy. The disease may rise to the nuclei of the bulb. Fibrillation precedes the atrophy. Reaction of degeneration is present to the degree of the completeness of the muscular loss. The extremities are livid and cold.

COURSE. The progress is so gradual, that it may be many months before one can be certain of the diagnosis, and then it may be two years more before the shoulders are affected, and several years more before the full degree of disablement is attained. The steps in the process may be marked by long remissions.

PROGNOSIS. Recovery is impossible, but the disease may stop at any point, and, after it is fully developed, the losses may be minimized by some of the unaffected muscles taking on new functions. If the bulb is implicated, death is the result.

FAMILIAL TYPE OF WERDNIIG AND HOFFMAN.

DEFINITION. A muscular atrophy which is familial, widespread, and fatal. Of a subacute or chronic invasion.

THE SYMPTOMS OF A TYPICAL CASE. An infant who has been very fat becomes weak in the hips and back, next in the arms, and then in the legs. Atrophy follows the weakness, and the bulbar muscles may be affected. It is fatal before the sixth year.

AGE. Begins from the sixth to the twenty-fourth month of life.

PATHOLOGY. It does not pick out muscles according to their representation in the segments of the cord, and thus is more like a dystrophy, than an atrophy.

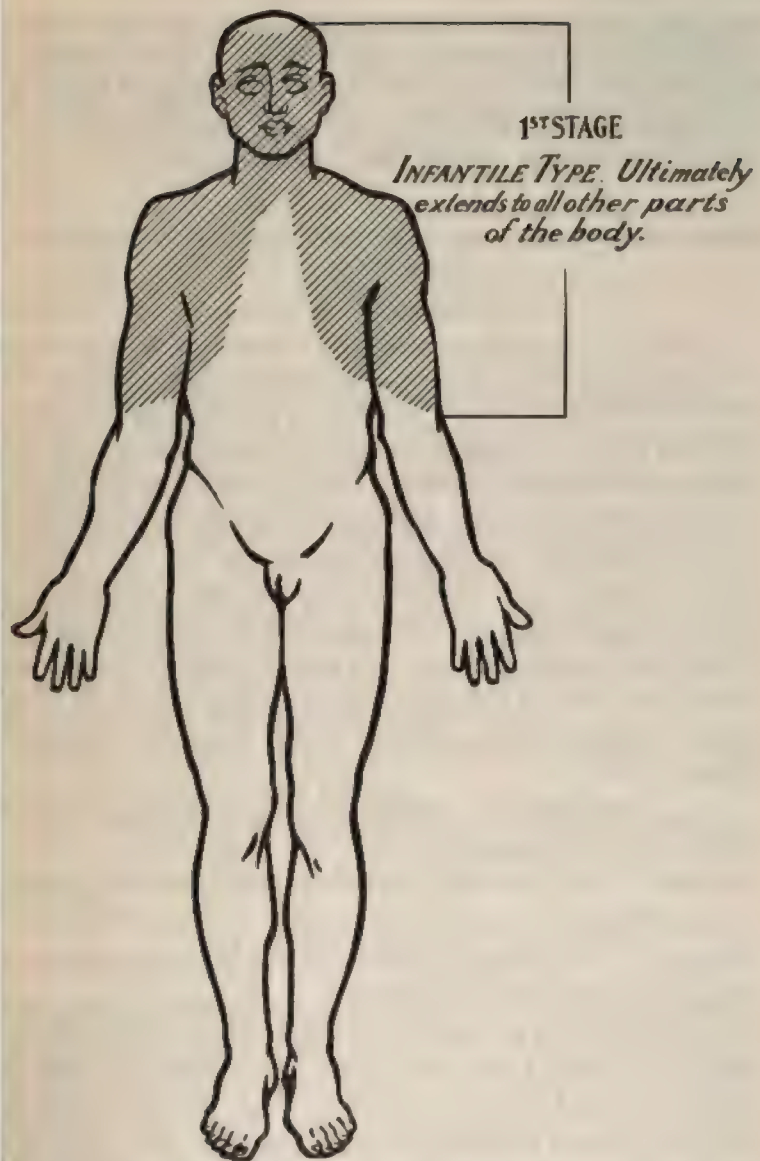


FIGURE 62.—MUSCULAR ATROPHIES.
(Modified from Mettler.)

SYMPTOMATOLOGY. The affected children are very fat. There is at first an atrophy, and subsequent weakness of the muscles of the hip and the back, which rapidly spreads to those of the arms, then next to the legs, and finally to the hands. Bulbar symptoms have been observed. It produces a complete helplessness.

PROGNOSIS. Death occurs before the sixth year.

PROGRESSIVE NEURAL TYPE—MYOSITIC TYPE—CHARCOT-MARIE-TOOTH TYPE.

(Peroneal Type.)

DEFINITION. An intermediate form between the atrophies and the dystrophies, since, at times, the nerves, while at others the muscles, are the point of initial attack. A simultaneous degeneration is, however, most common. It is called "Neural," from the fact, that from the spinal-cell to the muscle-cell, all parts of the lower motor-neuron are affected. It was called by others "Myositic," since the initial change might be in the muscle instead of the nerve, and by all it is called "Peroneal," since the initial weakness is always in the peroneal group of muscles.

THE SYMPTOMS OF A TYPICAL CASE. A person between the twelfth and thirtieth years shows an atrophy in the peronii, and acquires double talipes varus. After a long remission the whole leg atrophies, including the foot, and then the muscles of the thigh. Next atrophy attacks the hands and arms, finally leaving all four extremities cone-shaped. Electrical reaction is depressed, as are the deep reflexes; sensation is only slightly disturbed. Not fatal, but incurable.

AGE. The second half of childhood, and the latest limit of its appearance, is placed from the twentieth to the thirtieth year.

SEX. Shows no difference.

HEREDITY. It descends through the paternal line, which classes it with the atrophies. It has been seen in several members of a family, in which it resembles a dystrophy.

ETIOLOGY. It has been attributed to measles, and other infections, but it is certainly not syphilitic.

SYMPTOMATOLOGY. The muscles on the outside of the leg begin to waste, and gradually double clubfoot results, which may be the only condition produced for several years. The remission has been so complete at this point, that tenotomies have been performed for what appeared to be a completed process. The anterior, and then the posterior tibial muscles are wasted, and next, after years perhaps, the vastus externus, and then the adductors are also involved. After the tibials, the intrinsic muscles of the foot are atrophied. Now, after another halt, the hands, and then the arms, atrophy, and from the unequal losses "Claw-hand" results. The disease

may stop at this point, or may go on to a general atrophy. The atrophy is so selective, and follows such a definite order, that the limbs assume a cone-shape both in the legs and arms. Fibrillation is present, and a lowered electrical excitability, and often an early reaction of degeneration. Sensory disturbances are limited to a slight degree of numbness. Reflexes are decreased, and knee-jerk may be abolished.

COURSE. The progress is, as has been said, very slow, and years may be consumed in the process. It may stop at any point, and long intermissions are the rule.

PROGNOSIS. It is not apt to imperil life, as no vital centers are involved. The losses are apt to be permanent.

HYPERTROPHIC OR PSEUDO-HYPERTROPHIC PARALYSIS.

DEFINITION. This is a form of dystrophy, in which there is a hypertrophy, in some of the muscles at least, before the final atrophy.

THE SYMPTOMS OF A TYPICAL CASE. A young child seems over-developed in the legs, then becomes weak, and this may apply to all muscles, even to those of the tongue. The muscles of the back grow weak before those of the shoulder. Atrophy now ensues, and hypertrophy will be present in one part, with atrophy in progress in another. No reaction of degeneration, or reduction of deep reflexes appears until atrophy occurs. Progress is slow, attempts at cure futile, fatality not usual.

AGE. It may occur from the earliest to the latest periods of life, but is most frequent from the second to the twentieth years.

SEX. More males than females.

HEREDITY. It may be transmitted from one generation to another, through the mother, (atrophies through the father), and may also be familial, i.e., several members of a family being affected.

SYMPTOMATOLOGY. The change which first attracts attention is an enlargement of the muscles of both the thigh and the calf. It is at first considered a sign of perfection of development, but soon the child is found to fall easily, and to have little endurance, and the muscles, while enlarged, are found to be very weak. The tongue may also be the site of hypertrophy. At the same time, perhaps later, examination of the upper limbs will disclose an atrophy, and however distributed, the peculiarity of the disease rests upon an hypertrophy preceding a subsequent atrophy, and, also, that while one part is hypertrophic, another is becoming atrophic. The erectors of the spine are weakened at an early date, and before the scapular muscles show an atrophy, a line dropped from the cervical spine will fall clear of the buttocks,

on account of the pushing back of the shoulders in the attempt to keep erect by posture, rather than, as is customary, by the tonicity of the muscles of the spine. The hypertrophy is not absolutely confined to the muscles of the lower limbs, but is most certainly, and most prominently there. The general rule is this: hypertrophy is in the glutei, calf-muscles especially, and sartorius in the lower limbs, and in the deltoid, infra-spinatus, and triceps in the upper. Atrophy is in the pectoralis major, trapezius, serratus anticus major, latissimus dorsi, biceps, rhomboids, brachialis anticus, and later, in the quadriceps femoris, adductors, and then in the previously hypertrophied muscles of the lower limbs. The orbicularis oris, and palpebrarum (Starr says these are spared) atrophy and a mask-face may result in a late stage of advanced cases. The wasting does not affect the two limbs symmetrically, and the deltoid and the spinati do not waste along with the biceps and supinator longus, as in the atrophies. If the trapezius wastes, it is affected as a whole, and the upper part is not spared, as is the case in the spinal palsies.

The peculiarity of the atrophy produces peculiar deformities. The point of the shoulder drops from the atrophy of the trapezius, and serratus magnus, and the deltoid, and the scapula is loosened, and swings out from the wasting of the rhomboids, serrati, and levator anguli scapuli. Since the erectores-spinae are weakened the spine falls forward when the patient is in a sitting posture (lordosis), and its loss embarrasses the efforts to stand erect, or to rise.

Function is impaired in many ways. The patient can hardly sit erect without support; walking is waddling, with a tilting of the pelvis, first to one, and then the other side; stair-climbing is difficult or impossible; rising from a seat is only accomplished by pushing the body up by the arms, resting upon the knees. The typical losses are best brought out by the following test. Place the child upon its abdomen, upon the floor, and ask it to rise. It must first roll to one side, to get the knee under the body; supporting itself by this knee and the other arm, the other knee is gotten under the body; then one foot is placed upon the floor, and then the other; supporting the trunk with one hand, the other is placed upon the opposite knee, and the trunk pushed up to a vertical position, and the other hand on the knee of its side completes the process of walking the hands up the thighs, until the body is finally erect.

This wasting, and consequent paralysis may go on to any extent, but the muscles of the forearms and hands are usually spared. Contractures and deformities are common in the limbs, talipes equinus in the feet, and lateral scoliosis in the spine, from the unequal atrophy of the spinal muscles. Electrical excitability becomes reduced, as the muscular elements become unable

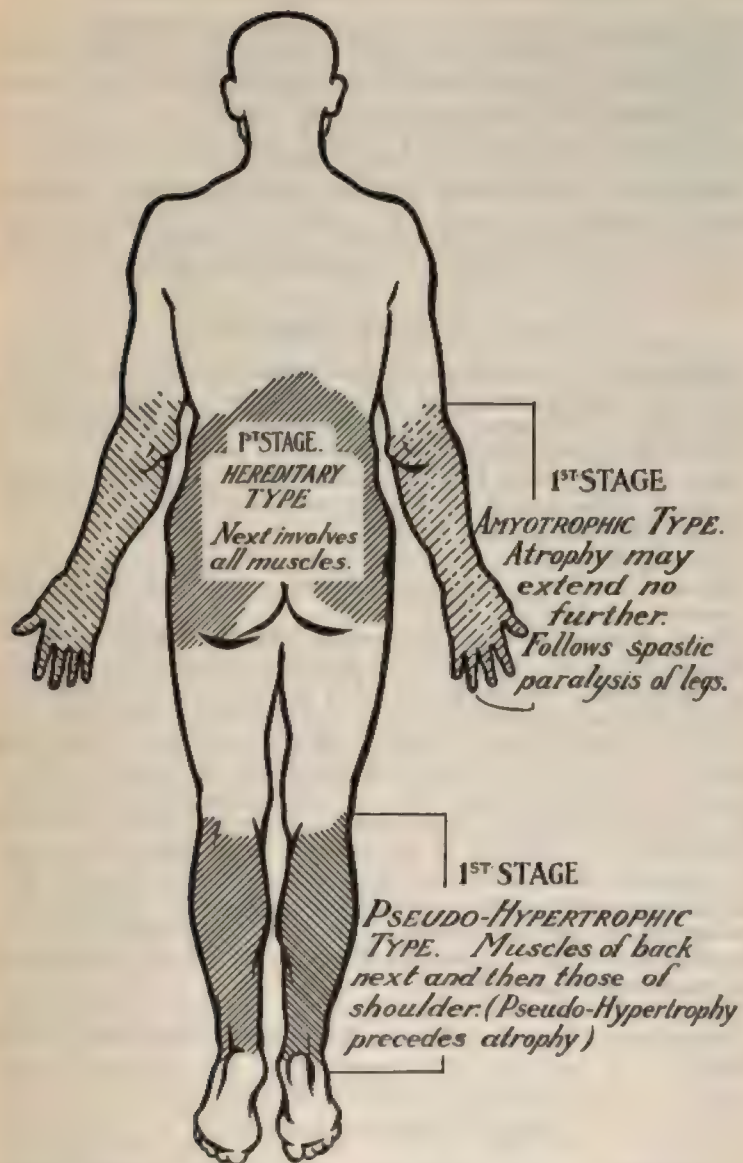


FIGURE 63.—MUSCULAR ATROPHIES AND DYSTROPHIES.
 (Modified from Mettler.)

to respond, but there is no reaction of degeneration, as in the atrophies. Fibrillation is absent. Reflexes are normal, as long as muscles have power. Sensation is not at all changed, unless the degeneration in the muscles is very rapid.

COURSE. Is so slow, as to be almost unnoticed at first, but in from four to ten years, the patient is helpless.

PROGNOSIS. The disease may not go to the extent of helplessness, but usually does, and cure is impossible. It does not tend to death, except from enfeeblement, which renders the person very vulnerable, especially to respiratory diseases.

ERB'S JUVENILE DYSTROPHY.

DEFINITION. A dystrophy which differs from the pseudohypertrophic type in the fact that it commonly occurs in youth, rather than in infancy, and that the initial point of attack is in the shoulder, rather than in the leg.

AGE. Early youth, twelve to sixteen, say most authors, but some extend the period to thirty-five, or even later. The majority are in the first-named period.

SEX. Equal.

HEREDITY. Not markedly hereditary, but is apt to be found in several members of a family.

SYMPTOMATOLOGY. The pectorales, trapezei, latissimus dorsi, and rhomboids are first involved, but the deltoid often escapes. Later the triceps and biceps atrophy, but the muscles of the forearm, except the supinator longus, and of the hand, escape. It may stop at this point, but if it does progress, it will next involve the muscles of the back, and then the muscles of the anterior part of the thigh, and then those of the calf, especially the peronei. In this disease, the atrophy is most often in the leg, while the initial hypertrophy is exhibited in the muscles of the upper extremity. It is selective, being mostly in the deltoid, if that is involved, and the supra-spinatus next. Sometimes the sartorius and gastrocnemius in the lower limb. The result is lordosis, talipes, and paralysis as in the preceding disease. There is no fibrillation, nor reaction of degeneration.

COURSE. Progress is very slow.

PROGNOSIS. Bad for recovery, but does not commonly cause death.

FACIO-SCAPULO-HUMERAL.

(Type of Landouzy-Dejerine.)

DEFINITION. It is perhaps the earliest type of the disease, finally ending in muscular atrophy, and is the most complete, this disease furnishing those museum curiosities known as "Living Skeletons." Peculiar in the atrophy of the face.

THE SYMPTOMS OF A TYPICAL CASE are an atrophy occurring first in the face, the patient being an infant, or a young child. The atrophy finally extends over the whole body, attacking the shoulder next, and then proceeding downward. The peculiarity is the age of the patient, the tapir-like mouth, and the apparent elongation of the neck, from the dropping of the clavicles. The progress is very slow and uniform, and life is not immediately endangered.

AGE. Most often begins in infancy, sometimes in childhood, and exceptionally, in adult life.

HEREDITY. This is directly hereditary, familial, and through the maternal line, like the other dystrophies.

SYMPTOMATOLOGY. Weakness and wasting are first manifested in the face, and most authors say that this wasting is particularly marked in the orbicularis oris, and palpebrarum, (Starr says that the eyelids escape). All agree that the muscles about the mouth, and the finer muscles of the lower face are attacked, and their function impaired. The levator menti, and the risorius are so affected, that the lips cannot be apposed strongly, and the zygomaticus is also atrophied. The process extends to the shoulder, and finally over the whole musculature of the body. The rest of the process differs in order of appearance only from the other forms of dystrophy.

With the whole body weakened by the atrophic paralysis of its muscles, the distinctive feature of this disease lies in the fact, that the mouth is tapir-like, the saliva dribbles, expression is lost, the cheeks puff out in attempts to talk, and it may be impossible to close the eyes completely. The neck seems to be elongated, from the dropping of the clavicles.

COURSE. Progress is so slow, and the atrophy is so uniform, that many people think that the patient is simply an instance of constitutional thinness.

PROGNOSIS. Hopeless for recovery.

TREATMENT. The cure of either an atrophy or a dystrophy is not considered possible, except in those cases of muscular atrophy which arise directly from a definite injury. The improvement, which sometimes occurs, seems to be more attributable to hygiene and local measures than to the action of remedies. This is, however, a debatable question, and careful prescribing sometimes reverses probabilities. Some cases have responded to adjuvants, and some are reported to have receded from symptomatic medication. The only broad difference in the treatment of the atrophies, and the dystrophies, is in diet. In the case of an atrophy, it should be rich in fats, and the contrary in the hypertrophies. In both there should be a moderate and constant exercise of the muscles to prevent the patients from becoming bed- or chair-ridden sooner than is necessary. Oppenheim alone differs in

advising the most perfect rest. All agree in the beneficial effects of massage and electricity, but warn against exhaustion of the muscles by too vigorous currents. Kellogg states that benefit has followed the use of a hot water pour on the spine, for a period of five to eight minutes, followed by a cold pour for from fifteen to twenty seconds, or an alternation of each for fifteen seconds, or alternate hot and cold compresses to the spine in the atrophies alone. The application of electricity should be carried out with great attention to technique. King advises, in atrophy, giving a moderately strong galvanic current through the spine. Place an anode, three or four inches square, over the diseased spinal segment, and the cathode over the sternum. A current of fifteen to twenty milliamperes should be used. After a little time, the anode should be moved its length, until the whole diseased area is successively covered in a treatment lasting about ten minutes. On the muscles, use a faradic current of tension, so as to make firm contractions as the electrode is moved rapidly over them, avoiding strong clonic contractions, which tire the muscle. Take from five to ten minutes, according to the number of muscles involved. Erb says: "Pass the moistened electrode over the surface of each affected muscle, keeping the electrodes close together, and using a moderate primary current. Do not interrupt very frequently. Treat only the muscles which react to faradism, and pay most attention to the most important muscles, and terminate the sitting by attention to any muscles which are threatened with an invasion of the disease.

THERAPEUTICS. The remedies with most evidence of efficiency are Argentum nit., Phosphorus with faradism, Cuprum, Plumbum, Gelsemium, Baryta, Mercurius. Others advised are, Zinc met., Arsenicum, Physostigma ven., Belladonna, Nux vomica, Strychnia, Lathyrus, Sulphur, and the Anti-psorics.

Physiological Medicine mainly relies upon Strychnia in some dosage. In the atrophies, Gowers says that by the mouth it is of no value, but the nitrate, hypodermatically, is valuable. He begins with a dose of 1-100th of a grain daily, cautiously increased to 1-5th. It has arrested very bad cases. Starr does not believe in it, but would rather give it by the mouth, 1-50th of a grain daily for four days, and then Arsenic 1-50th, the other three days of the week.

MYASTHENIA GRAVIS.

ASTHENIC BULBAR PALSY.

DEFINITION. This is a disease consisting of a progressive weakness of the muscles innervated from the pons and the medulla, but sometimes the muscles of the neck are the first ones initially affected. It finally amounts to a general paralysis of the whole body, which is purely motor.

THE SYMPTOMS OF A TYPICAL CASE. There is a gradual reduction in strength in the whole musculature of the body of a man in early adult life. The neck first becomes too weak to hold up the head, and then the eyelids fall as by their own weight, and the eyes cannot be held parallel causing diplopia. The muscles of the face and thorax are weakened, and then of the whole body. Reflex and electrical stimulation reveal the same readiness of exhaustion. This weakness may develop to any degree, but without actual atrophy. The disease is generally fatal in from two to four years.

DIFFERENTIAL DIAGNOSIS is from Progressive Bulbar Palsy, Cerebral Pseudo-bulbar Palsy, and Hysteria.

AGE. The majority of the cases occur in persons under thirty. Cases have been reported as young as the nineteenth year, or as late as the fifty-eighth. Tabulating all known cases up to a recent date, it is found that the average age of onset, in men, is thirty-five, and in women, twenty-four.

SEX. No difference.

HEREDITY. Not a factor.

ETIOLOGY. Since it is a disease of finally general distribution, it presumably is the result of the action of a toxin, although none has as yet been isolated. Generally, it is considered that this alleged toxin has a predilection for the lower motor neuron, and affects first those nuclei whose muscles are subject to the most constant use.

PATHOLOGY. No constant or efficient changes have as yet been discovered, unless the view is embraced that it is due to the persistence or changes in a persistent thymus gland, leading to a lymphoid infiltration of muscles. Such cases were reported as early as 1901, and, that these are comparatively unique, may rest upon the fact that reported autopsies do not involve an investigation of the mediastinum. Views which are more theoretical, have attributed it to changes in the sympathetic, and also in the muscles themselves.

SYMPTOMATOLOGY. Weakness in the muscles of the neck has been reported as the initial symptom in many cases. A case occurring in the practice of the author gives grounds for believing that ptosis and diplopia may antedate all other symptoms, and perhaps by several years. These ocular symptoms at first are transient, but finally return in a permanent form. Swallowing and chewing become very laborious, finally impossible, and continued speech readily exhausts the power of the muscles. The upper facial muscles also lose their efficiency, and the face assumes the mask-like look, which we find in paralysis agitans. After the involvement of the facial and cervical muscles, the muscles of the thorax become readily exhaustible, and those of the lower parts of the body follow in order, or are simultaneously weakened. Pure

cases will show no atrophy, but it has been observed as a complicating condition. After the disease has reached its full development, the patient will present the following picture. Ptosis has become so extreme, that the upper eyelids must be held up during the day by pieces of plaster, or some other mechanical device, and fastened together by similar means, to keep them closed during the night. Secretion of saliva seems to be exaggerated, and the power of swallowing is very deficient, and therefore the disposition of the saliva is a constant annoyance, and often a menace to life. In spite of this condition, it is said to be a point in diagnosis between myasthenia gravis and progressive bulbar palsy that there is no dribbling of the saliva in the first-named disease, since the corners of the mouth are not relaxed and patulous. The lower jaw tends to drop, and may have to be supported, and the head rolls loosely upon the trunk, so that a juremast is often essential to the comfort of the patient. While, as has been said, there is no atrophy, the distinctive feature is the ready exhaustibility of all the muscles of the body. In the earlier stages of the disease, muscles respond to volition, but become weaker and weaker with each contraction, until finally, the part is helpless. After a period of rest, they will respond again as before. In the later stages, it is practically a complete motor paralysis. This ready exhaustibility is just as true when we invoke electrical excitability. It is said not to be true of galvanism, but the first response to a faradic current is prompt and energetic, the next much less so, and finally there is a complete failure, no matter how strong may be the current. The sphincters remain intact, there is no reaction of degeneration, muscle-sense and coördination are never affected, there are no pains or other sensory disturbances, the special senses are preserved, and the reaction of eyes to light and accommodation are normal. Since respiration is a constant expression of muscular energy, the effects of exhaustion here are evident at all times, and measurement of the chest will show that between inspiration and expiration there will not be a difference of more than a quarter of an inch. The involvement of the pneumogastric nucleus affects also the rhythm of the heart, and there may be changes in the secretions. Mentality is generally entirely unaffected, and they may be able to transact business with success, but some cases have been reported, where there was mental excitement. Knee-jerk is exaggerated. Clonus is absent. There is speedy exhaustion of the special senses, as well of the skeletal muscles.

COURSE. There are remissions, and years may elapse between the initial symptoms, and the final definite onset of the disease, but this is not always true, as cases have been fatal in three months. Steady progression is not the rule.

PROGNOSIS. There is a tendency to death in from two to four

years. Grouping sixty reported cases, twenty-three ended fatally in an average period of eighteen months. Some cases have been reported as cured. Death occurs from inanition, choking, and inspiration pneumonia.

DIAGNOSIS. This depends upon the presence of a bulbar form of palsy, which is purely motor, and yet shows no atrophies, as a rule, nor reaction of degeneration. It is diagnostic also, that after rest the muscles are capable of strong contraction, but it is just as diagnostic that this power rapidly succumbs to the strain of repeated attempts at contraction. The point of attack is peculiar in being a ptosis, or weakness of the muscles of the neck. There are remissions of years, in the early stages. Fibrillation is absent.

DIFFERENTIAL DIAGNOSIS. Progressive Bulbar Palsy has the reaction of degeneration, fibrillation, and early atrophy. The saliva dribbles from the mouth. There is no ptosis, nor paralysis of the muscles of the neck, and there is certainly none of the muscular weakness of the whole body. It does not have definite remissions of long continuance. While neither have sensory symptoms, there are mental changes in progressive bulbar palsy, and a liability to apoplectic attacks.

Cerebral Pseudo-bulbar Palsy is the result of double, or alternate cerebral apoplexies. Mental changes are marked, and while mastication, swallowing and speech may be imperfect, the condition is due to spasticity, and not to weakness.

Hysteria may simulate it closely, since while neither have fibrillation, nor atrophy, they both may display muscular weakness. The weakness of hysteria is not so universal, nor has it the characteristic of ready exhaustibility to volition, and electrical stimuli. Myasthenia gravis has an absence of sensory symptoms, which are marked, and almost universal, in hysteria.

TREATMENT. In general the patient must have the best of surroundings, and the most complete rest, even from conversation. Gentle massage has its advocates, and general galvanization, but not to the extent of causing muscular contractions. The most constant care must be exercised to avoid choking, as the danger is always imminent, from the inability to swallow the abundant saliva. Small swabs should always be at hand, and tracheotomy may be necessary. Feeding can be successfully carried out by the nasal-tube, if swallowing becomes ineffective, or with the patient in the intubation position.

THERAPEUTICS. Phosphorus, Causticum, Cadmium sulph., Stannum, Belladonna, Veratrum, Lachesis, Hyoseyamus, Gelsemium, and Arnica have been advised on theoretical grounds for bulbar palsies, without much regard to type. Phosphorus seems best on the whole, unless we wish to go upon a basis of stimulation. In such case, Nux vomica, low, or Strychnia in any strength, and by any method seem the most promising. Stannum, high, has been

reported to have helped the choking attacks, and Hydrophobium, high, on pathological grounds, has been reported to be efficient by O'Connor. Tonics, Strychnine, and Veratrine are the physiological remedies.

MYOTONIA CONGENITA.

THOMSEN'S DISEASE.

DEFINITION. It is a disease characterized by a marked hypertrophy of the muscles, which is not followed by atrophy, but in some way induces spasm on attempts at voluntary motion.

THE SYMPTOMS OF A TYPICAL CASE are hypertrophy of certain muscles or muscle groups, with spasticity on movement, principally noticed in the muscles of progression. The whole body is thrown into contraction, but if it is possible to repeat the movements in spite of the contraction it finally gives way, and the muscles move normally until after another period of rest.

DIFFERENTIAL DIAGNOSIS. It is to be distinguished from Tetany, Para-myotonia congenita, and Pseudo-hypertrophic Paralysis.

SEX. More males than females.

AGE. While cases ordinarily present themselves to the physician in a period from the seventeenth to the twentieth year, careful examination of the history generally reveals the fact that traces of the disease have been evident from the earliest childhood. It has been seen to occur in a man of twenty-five years of age, but is most apt to become conspicuous about the age of puberty.

HEREDITY. It is often hereditary. It is generally familial. Twenty cases have been seen in four generations, and usually there will be found to be several cases in the same family, even if heredity is not traceable.

ETIOLOGY. Nothing is known. Fright has been stated to be a cause in one case. The theory of intoxication has appealed to many, on account of the diffused nature of the muscular injury. The patients are not noticeably neurotic, nor were the ancestors neurotic in hereditary cases.

PATHOLOGY. The only pathological change is a marked hypertrophy of certain individual muscles, or muscle groups, and on section, there has been found to be a multiplication of the nuclei in the sarcolemma, a change in the shape of these fibres from polygonal to circular, and an increase of the width of these fibres from double to four times their normal diameter; normal width being from 1-400 to 1-1000 of an inch. In this disease they have been found to be 1-150 to 1-500 of an inch. The transverse striation is always feebly marked.

SYMPTOMATOLOGY. Particular and diagnostic symptom is this: when voluntary contraction is set up in a muscle, it practi-

cally amounts to a tonic spasm, which will not relax for from ten to twenty-five seconds. This is on the initial contraction of the muscle, but, rarely, the second tonic spasm is greater than the initial one. The effect of this condition is, that after a person has been at rest and attempts any motion, these muscles become rigid, and a glass grasped by the hand is held there, further motion or relaxation being impossible. In generalized motions, like an attempt to walk, the whole body may be thrown into tonic spasm, and from the fact that we retain our equilibrium by innumerable compensatory contractions of antagonistic muscles, the patient may fall to the ground, incapable of any motion whatever. This condition of tonic spasm may be generalized, but as a rule, it is more strongly marked on one side than the other. The muscles of the face are usually free, but those of mastication are not uncommonly affected, and in some cases it is impossible to protrude the tongue, swallow or speak, until, by repeated trials, the spasm becomes relaxed. The finer muscles of the hand are almost always spared, so that while the patient cannot execute forcible movements of the hand with success, he can write with ease. This condition is most apparent after a long period of rest, after chilling, a cold bath, sometimes mental shock. It is minimized by continued exertion, by warmth, and, some have found, by moderate indulgence in alcohol. Erb stated, that electrical contractility is changed. There is the reaction of degeneration to galvanism, and the response to faradism is a slow and long-lasting contraction. There is a long, undulating wave starting from the cathode and going to the anode. The diagnostic point is the sluggishness of the reaction, the tonic character of the muscular contractions, and their long duration. If a strong faradic current is passed through a muscle, it contracts into rigidity, which lasts for twenty or more seconds, and if the muscle is sharply percussed, or even rigidly compressed, a knob-like swelling remains for some time. Respiration, defecation, micturition, or even coitus, may be embarrassed by the spasm. No sensory or mental change.

COURSE. This is slow, and the advent is hardly noticed, but is likely to become more evident about the time of puberty.

PROGNOSIS. Usually incurable, but there may be remissions, and one female case was better after marriage. It does not tend to death.

DIAGNOSIS. This rests upon the presence of an irregularly distributed hypertrophy of muscles, with tonic spasm on attempts at voluntary motion; decrease of this spasm upon continuance of the motions; long-lasting tonic contraction of a muscle after faradic stimulation, or from a blow, or from strong pressure.

DIFFERENTIAL DIAGNOSIS. Tetany occurs in those who are rachitic, the spasms begin in the hands, and Trousseau's sign is

found in this disease alone. The sign is this, that pressure upon the brachial artery will produce a spasm in the arms. This disease has premonitory symptoms, which are lacking in Myotonia congenita. They are numbness, prickling, and some local pain before the rigidity, and, in adults, some headache, and feeling of malaise.

Para-myotonia Congenita arises from the effects of cold, which causes a numbness and stiffness. This passes away, but some stiffness remains, and a degree of paralysis. The orbicularis palpebrarum and oris are especially involved. Muscular irritability is not increased.

Pseudo-hypertrophic Paralysis has peculiar deformities, contractures, and weakness. There is no tonic spasm on voluntary motion, nor on faradic, or physical stimulation of the muscles. At a later date it also shows atrophies.

TREATMENT. There is very little ground to hope for relief from remedies, but small doses of Veratrine are quoted by Jolly as beneficial. Others advocate massage, and educational forms of gymnastics. The individual must learn what will aggravate, and what will mitigate the spasm in his own particular case, but these measures are hygienic rather than medicinal.

FAMILY ATAXIAS.

DEFINITION. These are forms of degeneration of the nervous system which are principally evident in the cerebellum and the spinal cord, giving rise to some form of ataxia in young persons. While not always clearly differentiated, there is sufficient difference in the clinical picture to divide these cases into fairly well-separated groups, viz., Friedreich's Ataxia, Hereditary Ataxic Paraplegia, and the Heredo-cerebellar Ataxia of Marie.

THE SYMPTOMS OF A TYPICAL CASE. A young person progressively exhibits clumsiness, and mental dullness. In his later youth he has become incapable of following any avocation because he has no manual dexterity, he is uncertain in gait, and is also mentally deficient. The deep reflexes are lost in Friedreich's type, and increased in that of Marie.

DIFFERENTIAL DIAGNOSIS. The condition is to be diagnosed from Tabes Dorsalis, Chorea, Multiple Sclerosis.

FREQUENCY. The disease is rare in any one of its forms.

ÉTIOLOGY. This, like the pathology, applies to all the varieties, and will be described once for all; but other factors furnish grounds for the division into separate groups, and these will be made the subject of separate statement. Heredity, both direct and indirect, is prominent, and all the causes which are commonly known to develop a neuropathic condition in the individual are

sufficient to render the children liable to the degenerations of the nervous system which will produce some one of these forms of ataxia. The effects of a hereditary taint have been traced, in some instances for four generations, with a constant increase in the proportion of the children attacked, and a corresponding intensity in the symptoms of the succeeding victims. In some cases an exciting cause has seemed necessary, as only those children who have been subjected to the infection of typhoid, measles, scarlatina, rheumatism, or diphtheria (possibly) have been singled out to become ataxic.

PATHOLOGY. The common finding is a lack of developement in the cord, principally manifested in the posterior and lateral portions, although the cells in the anterior horns are wasted and deficient in number. In the type of Marie the cerebellum is especially degenerate, and the brain, in all cases, may be poorly developed. It is very clearly a lack of growth commensurate with the demands of increasing activity, and also of the power of resistance to functional demands upon the nervous structures. The cord and the cerebellum are smaller than normal.

FRIEDREICH'S ATAXIA.

AGE. Usually about puberty, but may arise in early childhood, or as late as twenty-five years of age.

SEX. Male cases are most common, but the inheritance is from the female side.

PATHOLOGY. The cord is diminished in size, it is degenerate, or undeveloped in all its tracts, and not only fibres, but cells also, are degenerated. The cerebellum is not always spared.

SYMPTOMATOLOGY. At puberty the patient is found to have become clumsy in his gait, and provokes comment among his companions on account of it, or he is constantly rebuked, at home, or in school, because he is continually dropping articles, or fails to write or to draw as successfully as his fellows. On account of this criticism he becomes bashful, and fails in mental grasp, apparently, and this may be the initial ground of complaint. If, however, he is carefully examined, no trace of mental dulling can be found at this stage. The musculature will now exhibit, on careful examination, an ataxia, most marked in the lower extremities, and there will be an apparent Romberg. If the test is made with both open and closed eyes, it will be found that the ataxia is as great under the one as under the other condition, showing that the fault is in the cerebellum. The result of this on the gait is that it is staggering and drunken, while in the spinal ataxia the gait is good in the early stages while the eyes are open, and the feet can be guided by vision. When the patient stands or walks, there is a nodding and wobbling of the head. Strength will be found to

be good, and therefore we can eliminate any paralysis. All tendon reactions are diminished and become abolished, and the knee-jerks soon become reduced to extinction, in a large majority of cases. The sphincters are unaffected in both this and Marie's type. The muscles become the seat of contractures, and the arch of the foot draws up, producing "pes cavus," while the contraction of the muscles of the foot produce "hammer-toe." All the toes may be affected. This combination of contractures is quite a point in diagnosis. Late in the disease, the same tendency to contracture irregularly manifested, produces scoliosis, most marked in the dorsal and lumbar spine. The involvement of the cranial nerves, in the later stages, is often shown by the presence of nystagmus, and speech becomes slurring, and the expression of the face is lost. The general muscular innervation is so poor that the patient becomes generally shaky; this may be evident in the face when talking. In the early stages there is apt to be a chorea, and it may remain permanently in certain localities. Electrical reactions are usually only depressed, but in a few instances some reaction of degeneration has been found. There may be some fibrillary twitching. Sensory disturbances are rare, but some cases suffer at first from pains, and later there may be a decrease in sensibility.

MARIE'S HEREDO-CEREBELLAR TYPE.

AGE. It develops after twenty, instead of before, as in the type of Friedreich.

SEX. Not a factor.

PATHOLOGY. This has many symptoms in common with Friedreich's type of ataxia, but autopsies show a very different localization of the degenerations, or mal-developements. The principal loss here is a mal-developement of the cells of the cerebellum.

SYMPTOMATOLOGY. Many symptoms are common to both varieties, but in Marie's type the tendon reflexes are exaggerated, and not diminished, but the superficial are not affected in either, nor are the sphincters. Chorea is more common and pronounced in this type, and optic atrophy and double-vision are peculiar to it, and Argyll-Robertson pupil may be present. While common in Friedreich's form, clubfoot and scoliosis are rare in Marie's type. All family ataxias occurring late in life tend to the cerebellar type, and have increased reflexes.

COURSE. The course of all is very slow, some having lived for from twenty to forty years, suffering only in locomotion, dexterity, and some mental impairment or non-developement.

PROGNOSIS. Most authors assert that no cases have been cured, but life is not threatened by the presence of the disease. Friedreich's type may go on to complete helplessness.

DIAGNOSIS OF FAMILY ATAXIAS IN GENERAL. This rests primarily upon the development of an ataxia in a young person, who can be proved to be free from a neuritis, the absence of the reaction of degeneration and of sensory symptoms, the presence of mental deficiency, with marked ocular and speech-defects, and "pes cavus," hammer-toe, and scoliosis, will be additional factors in its favor.

DIFFERENTIAL DIAGNOSIS. Tabes is like it in many respects, but the patient with hereditary ataxia is too young, his family history shows other cases, or marked neuropathy. He has no lightning-pains, no anesthetics, no bladder troubles, no visceral crises, no trophic disturbances, no loss of the muscle sense, and no Argyll-Robertson pupil, except in the type of Marie, in which the presence of increased deep reflexes would at once bar out the consideration of tabes.

Chorea. Since chorea may be an incident, or a complication, of both forms of family ataxia, the diagnosis must be upon broad grounds. In the history of a case of family ataxia, we find that the patient first became clumsy, shaky and ataxic, all depending especially, and often solely, upon some defect exhibited in the lower limbs. The choreic exhibits the characteristic movements abruptly and initially, and always most evidently in the upper limbs. Granting the condition to be atypical, or impossible of verification, there are symptoms present in the family ataxic which are absent from the symptomatology of the choreic. He has no nystagmus, nor scoliosis, no clubfoot, no hammer-toe, and the reflexes are not persistently diminished, or increased. In the Huntington's type of chorea the mind is initially and pre-eminently dulled, and there are forced movements and attitudes which we do not find in the family ataxias.

Multiple Sclerosis. This occurs in young people, and resembles in some particulars the type of Marie. It may also be hereditary, but in a well-marked case there are conspicuous differences. There is an intention tremor in the place of a general shakiness; the speech is staccato, instead of being slurring or elided; the limbs are spastic, instead of being ataxic, and there is an absence of swaying on attempting to stand erect.

TREATMENT. The only treatment recommended is an attention to hygiene, and an attempt at re-education of the muscles after the plan for the tabetic.

SYRINGOMYELIA.

DEFINITION. It is a slowly developing paralytic condition, characterized particularly by a loss of perception of pain and changes of temperature, while tactile sensation is preserved in the same areas. There is a condition of progressive muscular

and trophic changes in the skin, muscles, and joints. All these symptoms are due to a breaking down of tissue about the central canal, involving the cell nucleus in that region.

THE SYMPTOMS OF A TYPICAL CASE. A person slowly develops a paralysis of the lower limbs with wasting. The paralysis of the lower limbs may be spastic with an increase of the deep reflexes, while the wasting with diminished reflexes may be only evident in the upper extremities. Contractures follow the wasting form of paralysis, wherever it may be displayed. The essential symptom is that with intact sensibility to touch, the person will be found to have lost the perception of pain, and changes of temperature.

DIFFERENTIAL DIAGNOSIS. This is from Anterior Poliomyelitis, Progressive Muscular Atrophy, Tabes Dorsalis, Myelitis, Tumors of the Cord, Pachymeningitis cervicalis, Bulbar Palsy, Amyotrophic Lateral Sclerosis, Spastic Spinal Paralysis, Vasomotor Neuroses (like Acroparesthesia), Leprosy (from Morvan's type), Hysteria.

FREQUENCY. Not extremely rare, but not common.

SEX. More males than females.

AGE. Twenty-five to forty. Almost always before thirty, and never after forty, except from traumatism.

HEREDITY. Doubtful, but has occurred in three members of the same family.

ETIOLOGY. It has been noticed that an injury frequently precedes the disease, as the presence of a splinter of bone, or in another case under the observation of the author, it followed a crushing force, with resulting symptoms pointing to a hemorrhage in the cord. Syphilis has been found to produce cavities in the cord. Severe cases have developed in the absence of all exciting causes, and autopsies have disclosed beginning canalization, without being large enough to have been productive of symptoms. It is known that the glia tissue is thickest about the central canal, and also that the closure of the canal is sometimes incomplete, and that portions of the posterior septum are included, made up of embryonic cells. From all these data, the causes of this disease are considered to be due to a breaking down of embryonic tissue; the same process preceded by a growth of glia tissue; degeneration of glia tissue from vascular occlusion; or a destruction of otherwise normal tissue by a hemorrhage or laceration. Tissues, imperfectly organized from a fault in development, have been stimulated to abnormal proliferation by pregnancy, infections, or manual occupations involving strains to the spine, or elongation of it, which latter condition has been proven to be capable of increasing the volume of blood in the spinal cord.

PATHOLOGY. The disease usually begins in the cervico-dorsal segments, and is capable of an indefinite degree of extension both upwards and downwards, the former direction being commonly

the most extensive. The post-mortem appearance is very characteristic, since the cord appears normal, or only slightly enlarged, surrounded by normal meninges, but it tears easily, and feels soft. On rupture, a clear fluid flows out, and on examination the central canal of the cord is found to be enormously enlarged; it sometimes will admit the little finger, and its outline is very irregular. The enlargement may be interrupted, and instead of the cavity being formed by an enlargement of the canal, long tube-like cavities may be found entirely independent of it, the canal itself being normal. Such a cavity contains some masses of fibrin, proving it to have formerly contained blood (Hematomyelo-porus). At other times there will be anemic areas from compression of the spinal arteries, consequent upon a meningo-myelitis, or there will be evidences of a lymph-stasis, due to the pressure from vertebral caries. The typical condition is however an enlargement of the central canal, the walls of which will be the seat of some sort of gliomatous growth, which extends more or less into the surrounding gray matter. Most often it projects laterally, less often posteriorly, and least frequently into the anterior horns. It is usually not symmetrical in its extension. Occasionally it cuts off one-half the cord, giving rise to the Brown-Sequard syndrome of motor paralysis upon one side of the body, and sensory upon the opposite. The white tracts or columns are sometimes invaded, the posterior more often than the lateral, or the anterior.

SYMPTOMATOLOGY. The characteristic symptom of the disease is the fact that the perception of pain and temperature is lost, while ordinary tactile impressions are still appreciated in the same area. Muscle and joint sensation are present. The particular kind of loss may not be complete, since the appreciation of heat may be lacking, while that of cold may be still present, or the opposite. The patient is frequently unaware of his disability, and is therefore liable to injury in his daily occupation, and if a patient displays a degree of peripheral muscular atrophy, and shows evidences by numerous scars of having suffered repeated injury with little or no discomfort, it is

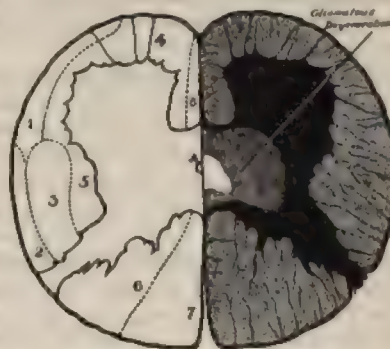


FIGURE 64. --SYRINGOMYELIA.

The gliomatous degeneration, arising in the walls of the central canal, invades the gray-matter of the anterior and posterior horns to a variable extent. This gives rise, first, to a loss of the perception of pain and temperature, and, to a variable degree, to paralysis and wasting with contractures.

presumptive evidence of the presence of syringomyelia. In the early stages the loss is usually in the hands, later in the shoulders and the upper trunk, and rarely may also be in the lower limbs.

TEST FOR TEMPERATURE PERCEPTION. Fill a test-tube with cold water, not ice, since extreme cold feels like extreme heat to most persons in health. Fill a second with water of such a temperature that it can be held upon the cheek for a minute without pain; carefully dry the outer surface of both tubes. Blindfold the patient, and place not just the tip, but a considerable surface of the tube on the skin area to be tested. Leave it in position for a few seconds, then try another area. Change from hot to cold, remembering to break the order, and do not preserve an alternation, and touch remote portions of the body from time to time, as a check.

TEST FOR PAIN. A pin is better for this purpose than a needle. Prick, alternating with touch. Change from one part of the body often enough to take the attention from the part. Make halts once in a while, and an answer when no stimulus has been given, will betray deceit, or inattention. Pain may be appreciated as a heavier pressure. The faradic brush is best of all.

The data from which a knowledge of the course of the fibres conveying the sensations of pain and temperature has been learned, has been gained from the post-mortem findings in this disease. We know that they enter by the posterior roots, go quite directly to the collection of cells located just postero-lateral to the central canal, and do not at once ascend in the white tracts, but run across to the opposite side by way of the anterior, or white commissure, and thus gain the column of Gowers, or antero-lateral tract in which they ascend. From this we know the lowest point at which these fibres are cut off, and can designate the particular segment injured, from charts which show what areas of the surface of the body depend for their sensory supply upon particular segments of the cord. In a late stage, tactile sensation may also be lost, but any one of the forms of sensory appreciation may exceptionally be preserved. Pain may be present.

The skin may show all sorts of vesicular, bullous or erythematous eruption, and these patients are particularly subject to numerous and extensive, but painless whitlows which are the result of repeated injuries to the tips of the fingers, which are unnoticed, on account of the lack of resulting pain. Whitlow is a lighter form, and Paronychia or felon is the designation of the severer type. This epidemic of painless whitlow is characteristic of that form of syringomyelia which is called "Morvan's Disease." The skin of the extremities is usually cold and sweaty, but at times there is a total absence of sweat. The nails are diseased, and may drop off. The muscles atrophy in a manner similar to

progressive muscular atrophy. It cannot be diagnosed from this condition until the sensory symptoms have appeared, since the atrophy is caused by the same lesion in both diseases; the result is the same if the sensory fibres going to the anterior horns are cut off, or if the cells in the anterior horns, lying toward the central canal, are destroyed. It is a sign that the cavity of the canal, or the gliomatous tumor which is a forerunner of cavity, has involved the trophic cells in the anterior horn. This wasting of the muscles is first seen in the finer muscles of the hand and thence slowly extends up the arm, to and involving the shoulder, and thence down to the trunk. It may begin in the muscles of the shoulder and scapula. These muscular groups are the seat of fibrillary twitchings, and show a partial reaction of degeneration. The ulnar and median groups are usually involved early, and from the unopposed action of the musculo-spiral group, there is over-extension of the wrist and hand, which tends to draw the hand into the curled-up position known as "Preacher's Hand." Spinal deformities are apt to occur, and we find scoliosis, or any other variety of spinal deviation, simple or combined, due to the fact that the asymmetrical distribution of the lesion puts an irregular tension on the spinal muscles. The face and throat muscles usually escape.

The bones are affected by an excessive fragility more often than in any other form of disease, and the joints are the seat of a painless swelling, and in this process the joint-surfaces disintegrate by a necrosis similar to that found in tabes. When atrophy of the soft parts is also present the fingers may drop off. The sphincters are not affected, unless their appropriate centers are directly attacked. Paralyses occur in connection with the atrophies in more than one half of the cases. As the disease slowly spreads downward, we find either a spastic paralysis, with exaggerated tendon-reflexes if the pyramidal tracts are involved, or an ataxic paraplegia if the posterior columns are invaded. In this latter case the tendon-reflexes are diminished. The spastic cases are also marked by the presence of some tactile anesthesia, but this will of course not be true if the pyramidal tracts are exclusively involved, yet their involvement does not take place without the great probability of some damage to the other structures in the vicinity. If the cavity-formation extends upward, we are pretty sure to see some degree of degeneration of the cervical sympathetic resulting from the destruction of the first dorsal segment, from which it derives its fibres. In such a case the palpebral fissure upon that side is widened, and there is a reflex iridoplegia, and while the pupil is not actually insensitive to light, the eye, when it has been shaded, will react very feebly. This loss is usually one-sided. The pupils may be unequal in size. Nystagmus is sometimes also present. A laryngeal paralysis may

occur, or some difficulty in swallowing, but the nerves of special-sense are usually undisturbed. It will readily be seen that there may be extensive and complete losses in the areas controlled by the nerves from the bulb, and the parts above it, when we recall that structures as high as the cerebellum may be practically hollowed out by cavity-formation, and that there may be a gliomatous tumor in the pons, and, owing to proliferation of the walls of the iter, there may be a resulting hydrocephalus, and resulting from that a compression of the brain-substance.

COURSE. The disease is a very chronic one, and may last for years. After a long term of steady progression, however, there may be complete cessation of further development, and, at any period, there may be intervals of long remission. On the other hand, at any stage of the disease, a paralysis may occur with an aggravation of all the existing conditions, such an accident depending upon the occurrence of a hemorrhage in the gliomatous new-growth, since, like all other tissues composed of embryonic cells, the newly developed blood-vessels are merely canals ramifying the friable tissues.

DIAGNOSIS. This disease is to be suspected, if we find a loss of the perception of changes of temperature and the presence of painful stimuli, but the negative element in the evidence must be insisted upon in an equal degree, viz., that in the same areas the tactile sensation must be preserved. This is necessary, since there are many conditions in which there is a loss of temperature and pain-sense, but with an equal degree of loss of all other sorts of sense-perception. If this peculiar sensory loss is found, either then, or as the disease progresses, we must also be able to demonstrate trophic disturbances of some degree in the skin, muscles and bones, and a muscular atrophy with paralysis. The combination of any two of these conditions, the sensory loss being always one of the two, is sufficient for a diagnosis. Since the lateral and the posterior columns are often invaded, or an entire section of the cord practically disintegrated by the degeneration of the glioma, we may find cases of apparent spastic or ataxic paraplegia, or transverse myelitis, where the presence of a dissociated anesthesia denotes that we are actually dealing with an extensive syringomyelia.

DIFFERENTIAL DIAGNOSIS. Anterior Poliomyelitis chronica and Progressive Muscular Atrophy (especially of the Aran-Duchenne type) cannot be diagnosed from syringomyelia until the peculiar sensory changes have become so evident, that one is able to satisfy one's self that the trophic changes are due to the invasion of the trophic cells by some process originating in the central canal, rather than from a primary disease in the anterior horns, or some peripheral portion of the nutritive apparatus. Any tactile impairment of appreciation proves that the disease

is not progressive muscular atrophy. Cases may exist for years without conspicuous sensory changes.

Typical Amyotrophic Lateral Sclerosis can readily be differentiated from syringomyelia from the fact that while both exhibit a paralysis, in the former the knee-jerks are never reduced, while they may be in the latter if the lesion is low down in the cord. The paralysis of the former is always spastic, while in certain localities the latter exhibits a flaccid and atrophic type. Bladder symptoms, scoliosis, paresthesias, unilateral bulbar symptoms, involvement of the trigeminal nerve, and nystagmus, are found only in syringomyelia. If the disease is atypical we must await the appearance of the peculiar sensory symptoms.

Spastic Paraplegias. The sensory changes of syringomyelia may not always be perceptible at an early stage, but they will not be continuously absent. Such a form of paralysis is apt to arise from a syphilitic meningo-myelitis, and in this we may get dissociation of sensation as well as in syringomyelia, but such a case will present different brain-symptoms. If the paralysis is of the Brown-Sequard type, the trouble is apt to be syphilitic, but if scoliosis, atrophic changes, and nystagmus (this last especially) are present, the case is syringomyelia.

Tabes. There is a great similarity at times when the lesion of syringomyelia is low in the cord. The disturbances in nutrition, even to disintegration of the joints, ataxia, loss of knee-jerk possibly, and the characteristic sensory changes may be common to both, and the diagnosis in such cases will rest upon some strong characteristics. If the upper parts of the body are relatively free from changes, except the presence of an Argyll-Robertson pupil, there is a loss of knee-jerk and Achilles-jerk, with disturbances of the sphincters and the sensory changes are the presence of spontaneous pains, and if a possible loss of the pain and temperature perceptions is accompanied by tactile losses in the same areas, the disease in all probability is tabes dorsalis. If on the contrary, there is increase of the knee-jerk, no disturbance of the sphincters, if there is scoliosis, if the atrophy is confined to the upper limbs accompanied by paralysis proportionate to the atrophy, and there is a loss of the perception of pain and temperature over large areas, while the tactile sense is normal in those areas, the disease is most certainly syringomyelia.

Myelitis. In either the transverse or disseminated form, this may resemble syringomyelia. Myelitis, however, usually affects the lower limbs, while syringomyelia is more often in the upper; its losses are more unequal upon the two sides, and the peculiarity of the sensory loss is generally diagnostic.

Tumor of the Cord. Tubercle, gumma, and glioma are the principal varieties. The first two are possibly diagnosed from their history. A more rapid invasion of an area limited by two

or three segments of the cord, instead of five or six, and a steady progress indicate a tumor. If a tumor is larger than has been suggested, it is liable to produce the symptoms of an extensive compression myelitis, and there will be paresthesias, pain, involved sphincters, and bedsores. The Brown-Sequard syndrome is more probable in tumor than in syringomyelia.

Pachymeningitis Cervicalis Hypertrophica. This disease runs a more rapid course, there is local tenderness in the spine, the head is kept in a fixed position, there are root-pains of a very pronounced character, extending to the upper chest, the shoulder and the arms, there is less atrophy, but a more pronounced reaction of degeneration. The anesthesia also is for all forms of sensation, and it is distributed in smaller areas.

Bulbar Palsy. Syringomyelia may develop bulbar symptoms, but it is long after the arms have shown their peculiar atrophy and paralysis, and the legs have become somewhat spastic. Bulbar palsy begins in the bulb, and all the symptoms common to the two diseases are later additions.

Caries of the Spine. At a late stage of this disease, we may find symptoms which remind us of a syringomyelia, but it has been the outcome of a compression or secondary myelitis. The sensory disorders are rarely of the proper type, and the history of tuberculosis, or some of its indications, can be discovered, and the X-ray discloses changes in the vertebrae.

Leprosy. This resembles that type of syringomyelia called "Morvan's Disease," but neither in the cutaneous lesions, nor in the cord post-mortem, can the bacillus be discovered in syringomyelia. Another point of difference: in "Morvan's Disease" the plaques are upon the upper extremities alone, while in leprosy they are also upon the face and legs.

Hysteria. This may show the sensory losses peculiar to syringomyelia, as it does those of other diseases but the stigmata of hysteria are apt to be present. Failing these, the patient is apt to be younger, of the female sex, the disease comes on over night, or in a very few days, and some emotional strain, either sudden, or prolonged, is the evident cause.

TREATMENT. The disease does not seem amenable to treatment, when we refer to a restoration to former health, but there are many conditions which must be cared for, and attempts must be made to aid nature in the halts which sometimes occur, or in the total cessations, which are a rarer contingency. Hygiene and massage will conserve the muscles, and electricity will aid in this, and will also benefit the atrophies, whether they be general or local. The possibility of favorably influencing the resolution of the whitlows by the negative galvanic electrode (stable) should also be kept in mind. The areas of analgesia should be protected from harm, and the slightest injuries should be treated

with the most rigid aseptic precautions. Use apparatus for the scoliosis.

THERAPEUTICS. There is little to guide us here, but for all atrophic conditions Phosphorus 6th would seem best indicated. Some have advised Plumbum met., Cuprum met., Zinc met. (upon the results of poisoning by the metal), Arsenicum, Fluoric acid, Argentum nit., Physostigma venenosa, Belladonna, Nux Vomica and the Anti-psories. For the whitlows O'Connor speaks of Dioscorea, Natrum sulph., Silicea, Sulphur, Hepar sulph., and Calcarea, but the whitlow of syringomyelia is painless, while the symptomatology of all these remedies is so very rich in sensory symptoms, that their use must be entirely experimental.

BULBAR PALSY.

PROGRESSIVE BULBAR PARALYSIS: PROGRESSIVE LABIO-GLOSSO-LARYNGEAL PARALYSIS: POLIO-ENCEPHALITIS
INFERIOR CHRONICA OF WERNICKE.

DEFINITION. It is a degeneration of the motor nuclei in the bulb, which give rise to the nerves which innervate the muscles of speech, of facial expression, of phonation, deglutition, and finally of respiration. This results in a slowly developing paralysis of these muscles, and the more or less complete abolition of their functions.

THE SYMPTOMS OF A TYPICAL CASE. A person in late adult life will develop a slurring speech, in which some consonants are poorly pronounced. This disability will increase, and it will be found to depend upon a paralysis, with wasting of the tongue and lips, which will become very marked in the entire lower portion of the face. Apoplectiform seizures will be an incident of the later stages of the disease.

DIFFERENTIAL DIAGNOSIS. To be differentiated from Dysarthria, Tumors, or Hemorrhage of the Bulb, Multiple Sclerosis, Amyotrophic Lateral Sclerosis, Bilateral Facial Palsy, Myasthenia Gravis, Infantile Bulbar Palsy, Thrombosis or Embolism of the Bulb, and the inflammations of this region, which are termed Polio-encephalitis superior or inferior, according as they are located above, or include the nucleus of the Facial nerve.

FREQUENCY. It is a rare disease.

AGE. The period of active adult life from thirty to fifty, but while very rare before forty, has been known as early as thirty, and as late as sixty.

SEX. Both equally.

HEREDITY. It is not one of the diseases in which heredity seems to be capable of demonstration.

ETIOLOGY. The actual cause is unknown, but cases have been known to follow cold, exposure, toxemias (Hoffmann quotes a case from lead poisoning), and it has seemed to have resulted from hereditary syphilis. Over-use of the muscles of this region has been given as a cause. A case quoted was from the blowing of wind-instruments. Such a result indicates a neuropathic tendency, and stamps the underlying cause as a localized pre-senility. The exciting cause may be any form of over-strain, either general or localized.

PATHOLOGY. Speaking broadly, it is a degeneration of the nuclei in the medulla of the motor cranial nerves. Palsy of the hypoglossal would paralyze the tongue, of the spinal accessory would paralyze the trapezius in the upper third, and the sternocleido-mastoid muscles. The degeneration of the glosso-pharyngeal would paralyze the pharynx, while the degeneration of the fibres originating in the accessory nucleus, but continuing in the pneumogastric, would paralyze the larynx. The paralysis of the pneumogastric affects respiration and heart's action. The degeneration of the facial would paralyze the superficial muscles of the face, the frontalis, platysma myoides, stylohyoid, and the posterior belly of the digastric, but since only one of the several nuclei of origin is the seat of degeneration, the loss is diagnostically limited to the muscles of the lower face. The loss of power in the lower part of trigeminus, or tri-facial, would paralyze the muscles of mastication. Since the nourishment of the muscles depends upon the integrity of the cells in these same nuclei, we shall find first *fibrillation* of muscles, and then atrophy of them. This is analogous to any spinal degeneration involving the cells of the anterior horns, for the nuclei of motor cranial nerves are the analogue of these cells in the cord. It is like an amyotrophic lateral sclerosis, but lacks the spinal symptoms. Sometimes they are present in the arms, and hence it has been classed by some with this disease, and considered simply as a bulbar type. They are not always equally affected, and in some cases losses are erratic. Tooth and Turner found in one autopsy that there was no lesion of the 9th, 10th or 11th, but that the motor part of the 5th, a part of the 7th and the 12th were degenerated. In general the hypoglossal is most frequently degenerated, next the accessory part of the vagus, then the glosso-pharyngeal, then the motor part of the 5th, and lastly the facial. From this distribution we see that the functions of speech, deglutition, mastication, and facial expression are affected. The ganglion cells of these nuclei gradually degenerate, showing this condition by the fact that their dendrites and axons fall away. The condition affects other structures within the area, and therefore nerve-fibres passing to higher or lower levels are also degenerated. This is particularly true of the pyramidal fibres. It affects also the intra-muscular fibres, and

the muscles themselves. No sensory changes occur, as the sensory nuclei and fibres seem to resist the process.

SYMPTOMATOLOGY. The clinical symptomatology demands a description, from the fact that there is a diagnostic order in the evolution of the symptoms, and because some symptoms occur, or are lacking, without apparent reason. Briefly: there are disorders of speech, then of deglutition, next of mastication, then of phonation, and at last the respiration, and the action of the heart are affected. The first symptom is paralysis of the lingual and facial muscles in succession, which condition always denotes a degeneration in the cells of origin, or of the fibres of the nerves in continuity, and this paralysis is always foreshadowed by a weakness of the muscles. It follows therefore that the patient shows fatigue from a long conversation, and so may be an earlier means of manifesting the weakness than spoken speech. Next it is noticeable that d, t, r, u, s, ch, and i, are indistinctly pronounced, showing that the tongue is not correctly and rigidly apposed to the teeth, and the roof of the mouth. Before atrophy can be detected, on examination the tongue will be found to display a flickering movement in the separate fibrillae of the muscles (fibrillation), and at length it will be too weak to be capable of protrusion. This preliminary fibrillation is true of all muscles afterward atrophied. Next there is weakness of the orbicularis oris, and we shall find that p, w, f, m, o and u, will be pronounced indistinctly, on account of the weakness of the lips, and fibrillation, and trembling, will later be seen in them. Then the voice will acquire a so-called nasal twang, on account of a weakness of the palatal muscles. A slight bilateral weakness is much more disabling than a far greater unilateral palsy. From the same reason liquids, and sometimes particles of food, will escape through the nose. At this time we are apt to hear complaints of the only variety of sensory symptom present. There will be some degree of pain in the back of the neck, or an occipital headache. This combination of losses produces what is known as Bulbar speech; an enunciation which is blurred, nasal, and run together, in the attempt to complete a sentence before being overtaken by exhaustion. The vowels alone are perfectly pronounced, except occasionally o, and u, which require the perfect apposition of the lips. Such a condition is Dysarthria.

By this time, the loss of power in the facial muscles has become so pronounced, that the play of expression in the *lower* face has been impaired, and in time is wholly lost. As the lips become feebler, the saliva dribbles from the corners of the mouth, both from the weakness of the lips, and also from the irritation of the chorda tympani of the facial. Without apparent reason the ocular muscles are very rarely implicated, and the zygomaticus, the elevator of the upper lip, the orbicularis oris, and the frontalis are

exempted. At the same time that the palatal muscles show enfeeblement, the pharyngeal muscles begin to weaken, and there is difficulty in deglutition. At about the same period, the muscles of mastication, which are innervated by the motor branch, coming down with the third branch of the 5th, show degeneration, and chewing is performed imperfectly and with difficulty. Particles of food are apt to lodge between the cheeks and the teeth. When at length the larynx weakens in its turn, the vocal cords become paralyzed, and the voice becomes hoarse and toneless, and finally altogether fails. This is Aponia.

Lastly, degeneration of the nucleus of the pneumogastric causes respiration to become feeble, and this combined with the inefficiency of the larynx, produces spasms and wheezing on inspiration. From the same nuclear degeneration the heart's action becomes irregular, and is accelerated, the pulse rate being from 120 to 140. The patient is now apt to inspire the mucus which is so freely secreted, not only producing choking, but, in addition, it is apt to produce asphyxia, or an inspiration pneumonia which is a common cause of death.

Atrophy after a time becomes very pronounced, especially in the muscular structure of the tongue. This ensues to such a degree that the mucous membrane lies in folds upon the tongue, and the whole organ becomes thin and colorless, and lies quivering upon the floor of the mouth. When the contiguous pyramidal fibres are also degenerated, we shall find some amount of spastic paralysis in the arms, and less often, in the legs, and under such conditions, and then only, we shall be able to elicit a jaw and malar jerk. So long as there is any muscular tissue there will be some electrical reaction, but faradism first fails, and next there is noticeable slowness in reaction to the galvanic current, and next the anodal closure will equal, or even surpass in effect the cathodal closure. This is the reaction of degeneration. Finally, the muscles fail to react to either current. Late in the disease we may see convulsions of an epileptiform type followed by mental confusion, but the mind is usually clear. The patients are apt to be sorrowful, and the emotions are easily excited. The sorrow is probably from the fact that they are mentally acute enough to realize the seriousness of their condition, but such a mental state always follows lesions affecting the pons. Some writers on the contrary insist that the patients are actually good-humored, in spite of the disease, although all agree that they are emotional. There is a general impairment of health, and patients show a progressive general emaciation, due to a lack of appetite inseparable from their invalided condition, and to inability to masticate and swallow sufficient food. The pharyngeal and laryngeal reflexes are generally lost, but not invariably. The deep reflexes are usually exaggerated.

COMPLICATIONS. The disease is associated at times with some degree of Progressive Muscular Atrophy, and Amyotrophic Lateral Sclerosis.

There is a form which has been termed acute apoplectic, which arises from hemorrhage, softening after thrombi, more rarely after emboli of the vertebral, and basilar arteries. It may be simultaneous with similar conditions in the brain.

COURSE. The disease is insidious in onset, slow in development, for a time making no progress, but remissions are rare and short, and there is never any conspicuous recession. In a few cases the onset has been acute, but only in an unusually rapid evolution of the speech defect, for even in such cases the remaining symptoms evolve at the usual rate.

PROGNOSIS. The disease rubably terminates in death in a period of one to three years, although some cases have been slower, while others have been fatal in the first year. The cause of death is either inanition, pneumonia from inspiration of mucus or food into the trachea, or it arises from some intercurrent disease to which such individuals can offer very slight resistance.

DIAGNOSIS. When the case has come to an advanced stage there is no particular difficulty in the diagnosis, but if the disease picture is still incomplete there may be reason for doubt. In the early stage, however, no matter how inadequate the history, certain changes are sure to be present which strongly suggest the true nature of the malady. There is even then some grade of paralysis of the tongue, and it is quite diagnostic that it is bilateral and progressive, and that invariably there is some degree of paralysis of the lower face and lips accompanying it. The patient cannot whistle, even if the anterior nares have been previously closed, nor can he blow out a candle. He cannot elevate the tip of the tongue, nor protrude it, nor place it in the sides of the cheeks on request. If the finger and thumb are placed between the lips their separation is effected with little force. Even when the reaction of degeneration has begun, some response will remain so long as any muscular tissue persists. Faradism first fails, then comes the reaction of degeneration, and then all response is finally lost. Slowness of response is seen in the early stages. Sensation is unimpaired. Patients are in later middle life. Slowness of onset is a diagnostic sign, and of great importance. Of prime diagnostic importance is the *order* of invasion, and that the paralyzes shall be bilateral, and marked by atrophies, and the reaction of degeneration, and shall be only such as could be predicted by the degeneration of the nuclei of the motor cranial nerves.

DIFFERENTIAL DIAGNOSIS. Dysarthria from other causes must also be excluded. If it is due to an anatomical fault in the vocal mechanism, either congenital or acquired, it will be revealed by a careful examination. If it is the result of the action of some

corrosive poison it should not be beyond discovery. If it is the late result of diphtheria, tonsillar inflammation, or some other infection, there is apt to be a history of the causative disease. If this is not discoverable, its localization, the associated paralyses, or the lack of them, the rate and manner of onset, the electrical reactions, the presence, or absence, of atrophies, and the age of the patient are all means of differentiation. A unilateral paralysis of the vocal cords resulting from a lesion of the recurrent laryngeal nerve makes the voice hoarse, but does not interfere with the articulation of consonants.

Infantile Bulbar Palsy. Some writers state that infants occasionally suffer from bulbar palsy, but there is a sharp line of demarkation between the adult and the infantile conditions. Progressive bulbar palsy is slow in onset, since it is a degeneration, while the Infantile variety is rapid, since it is an inflammation of the gray matter of the bulb, and is not always fatal. It is a poli-encephalitis, and its rate of onset is diagnostic. There is a slow variety which is more confusing, as its rate of onset is similar to that of the adult form, but it may be differentiated clinically, from the fact that it attacks the upper face, and confines itself most distinctively to that region, which is peculiarly the region spared in true bulbar palsy; deglutition is not affected, but there is ptosis. Moreover, such children are degenerate. They are the product of consanguineous marriages. Tenderness of the bones of the face on percussion is one of the signs of such degeneracy. The adult form is a degenerative disease of the bulbar nuclei, but the infantile is a nuclear atrophy.

Pseudo-Bulbar Palsy. This has many points of similarity. The site of the lesion is, however, in the cortex of the brain, and it is the result of a separate lesion in the two hemispheres; but while it does result finally in a bilateral paralysis of the tongue, hypoglossal, and of the lower face, yet it is not bilateral at the onset. There is no atrophy, the lingual muscles will react to faradism, and there is never a loss of the pharyngeal and laryngeal reflexes. While the upper limbs are at times spastic in progressive bulbar palsy, the lower limbs are the seat of a distinct double hemiplegia in pseudo-bulbar palsy.

Tumor. Tumors of this region may be near to as well as in the bulb. Effects similar to progressive bulbar palsy may be produced by a tumor pressing upon the bulb, or by a thickening of the bone about the foramen magnum, or of the odontoid process of the axis, but these symptoms are rarely bilateral and difficulties of deglutition are the most pronounced effect. If a tumor is situated in the bulb it is slow in progress, and may produce many of the symptoms of bulbar palsy, but they are rarely bilateral in distribution, the symptom-complex is irregular and incomplete, there are sensory changes, and symptoms of intra-cranial pressure

are also present. The classical tumor symptoms are modified somewhat when it is located in the medulla. Vomiting is very prominent, vertigo is persistent, and cerebellar in type, while the usual optic neuritis is often absent. The difficulties of diagnosis, however, may be appreciated when we note that Wilks mentions a case where no symptoms at all were developed during life.

Hemorrhage. This might produce all the symptoms, but it is of rare occurrence and is almost always fatal when it occurs in this locality. The apoplectiform character of the onset should immediately show that it was a sudden accident, and not a gradual process of degeneration. Unconsciousness is usual, but not invariable.

Softening. The other vascular accidents are Thrombosis and Embolism of the basilar, vertebral, and anterior spinal arteries, of which the vertebral and its branches are the most frequent site. The result of both these conditions is softening, in which some of the cranial nerve-nuclei are sure to be involved. The result will be some degree of palsy in the muscles innervated from the bulb, but the onset will be a matter of a few days instead of months, and the distribution will be erratic, and not so purely motor as in bulbar palsy. Unconsciousness is rare. Miller reports a case, and quotes others, showing that thrombosis in the region of the bulb is of a definite type. The patients do not lose consciousness, but are apt to become suddenly helpless in the lower limbs, and to show a paralysis of the vocal cords on one side, and ataxia in the same side of the body. The same side of the face and the opposite side of the body show heightened sensitiveness to heat and pain. In other words there is a paralysis of the parts of the body spared in bulbar palsy, and in addition a crossed hemianalgesia (loss of pain perception).

Multiple Sclerosis. This has been a confusing possibility at times, since both diseases have a speech defect, the diagnostic differences between the two varieties being less easy of detection than is usually stated. In passing it is best to remind the reader that later investigation has shown that cases of multiple sclerosis are seen without the speech defect which has been esteemed diagnostic. The typical defect is that the patient speaks in a staccato manner, explosively, because the form of paralysis affecting these muscles is marked by a rigidity. In bulbar palsy, on the other hand, the fault is a slurring, a clumsiness, as well as a weakness of articulation. In multiple sclerosis the facial palsy is not a bilateral weakness, but a stiffness with weakness, and is unilateral. The extremities also suffer from a stiff weakness which is bilateral, as they rarely may in some forms of bulbar palsy, but even then the sufferer from bulbar palsy does not display the intention tremor, nor the nystagmus of multiple sclerosis.

Amyotrophic Lateral Sclerosis. If to the bulbar symp-

toms are added the symptoms of a degeneration of the cells of the anterior horns of the cord (shown by a spastic paralysis of the lower limbs, with wasting, fibrillation, the reaction of degeneration, without sensory disturbances), we are dealing with a more general disease of the same type as bulbar paralysis, but designated by another name for purposes of differentiation only. In this case, and not in the typical bulbar palsy, the wasting and paralysis of the face, mouth, and throat will be preceded by a spastic stage, and the jaw and malar-jerk will be found.

Myasthenia Gravis. Here we find early ptosis, paralysis of the orbicularis palpebrarum (the paralyzes of bulbar palsy are confined to the lower face), as well as of the other muscles from the bulbar nuclei, weakness of the muscles of the neck, so that the head rolls about, which is late if ever in bulbar palsy. There is greater and more prompt muscular weakness in myasthenia gravis, and the knee-jerks are never exalted, but show the peculiarity of ready exhaustion on repeated exhibition, as do all of the other muscles affected. There is no reaction of degeneration, but instead this diagnostic feature: the response to all forms of galvanism diminishes progressively as it is repeated, but after a period of rest is found to be as energetic as at first. Remissions and recessions are frequent, and cure is not unknown.

Polio-encephalitis of Wernicke. Polio-encephalitis is an inflammatory condition which has been divided into superior, confined to the eye-muscles, and an inferior type which produces symptoms of bulbar palsy; but the onset is acute, and a certain per cent., less than half, of these cases recover. There is a simultaneous involvement of the nuclei, instead of a gradual extension from one to another. It is common also for structures other than the nuclei to be involved, and so it is usual to find various forms of alternating hemiplegia, and hemianesthesia, i.e., a loss of muscular power on one side of the body, and of sensation on the opposite one, or a general paralysis may be the result.

TREATMENT. This falls into the two divisions of adjuvant and hygienic, and therapeutic. The hygiene should be of the most perfect kind, and over-exertion, particularly of the vocal organs, should be strenuously forbidden. The food should be of the most nourishing kind, there should be plenty of fresh air, and the nutritional effect of massage, both general and local. Hot baths and packs followed by the cold douche or spray are of use in toning up the general circulation, and any shock should be antidoted by vigorous rubbing after the bath. When in the process of the disease swallowing has become difficult, great care should be exercised to prevent the abundant mucus from falling down the trachea, and causing choking and pneumonia. This may best be obviated by having at hand a number of long swabs of absorbent cotton which should be used in a routine manner, in preventing

great accumulations of mucus in the mouth, and all attendants should be sufficiently trained to clean out the pharynx on a moment's notice. When the failing power of deglutition has begun to affect nutrition, the stomach-tube, or preferably the nasal-tube, should be employed, and each feeding should be followed by four ounces of water. When the stomach has become intolerant, feeding must be by rectum, and nutritive enemata should be followed by six ounces of normal salt solution to combat thirst.

Electrical Treatment. This should be tried for a short time, but not if the case comes into one's hands with a well-marked reaction of degeneration, or if this comes on in the course of the disease. In no case should the treatment extend over ten minutes in all the areas combined, since degeneration in the affected nuclei is hastened if they are stimulated to exhaustion. That this has been produced, is shown by a demand by the muscle of a greater current to produce contraction. The medulla as a whole may be stimulated by passing a galvanic current from one mastoid process to the other, with electrodes about four by four inches. Vertigo may ensue if the current is strong. Another method is to place the positive electrode over the base of the brain, and the negative over the motor points of the lips, the masseter muscle, and the tongue. This current is preferably a galvanic one, making from five to ten interruptions over each muscle. Stimulation of the deglutition reflex is valuable, since difficulty in swallowing is a vital loss. This may be accomplished by the faradic current with one pole on the nucha, and the other over the poma Adami, or by the galvanic positive on the nucha, and the negative in front. A more exact method is to place a small electrode upon opposite sides of this projection, and pass a current between the two. Galvanization of the sympathetic is accomplished by placing the negative at the clavicle, close to the spine, and the positive at the nucha. This will also alleviate the stiffness of the neck. A general treatment of the whole body with faradization, or by the static breeze, has also been advised. From any form of electrical treatment we can, at the most, hope only for a retardation of the degeneration.

THERAPEUTICS. The remedies advised for this condition have been based upon clinical reports of the relief of various isolated symptoms without sufficient data, in most cases, to inform us whether the symptom-complex was pseudo-bulbar palsy, which is a cortical lesion, or a bilateral one of the cranial nerve tracts, or true degenerative bulbar palsy, or myasthenia gravis. Since pseudo-bulbar paralysis is not accompanied by atrophy it is evident that remedies marked by that symptom are inapplicable, while the contrary is true in the case of the degenerative form, which is especially distinguished by the presence of atrophy.

Cure is not possible, but there is evidence that some of the symptoms may be benefitted for a time. Remedies selected upon toxicological grounds have not proved efficient, those based upon pathological changes seem to be rather more successful, but our main indication should be symptomatology. Manganese binocide, and Cadmium sulph. are the toxicological remedies, while Mercury is pathologically suggested. Arnica, Argentum nit., Baryta carb., Causticum, Lachesis, Nux vomica, Phosphorus, Plumbum, Stannum, and Veratrum viride should be studied for a similimum.

CHAPTER VII

DISEASES AFFECTING THE BRAIN AND ITS ENVELOPES

CEREBRAL PACHYMENINGITIS.

DEFINITION. This is an inflammation of the dura of the brain, and is a much more rare affection than an inflammation of the pia. It most commonly affects the thick external layer alone, and it is then termed pachymeningitis externa, and less often the thin internal layer, when it is called pachymeningitis interna. It may be acute, or chronic, purulent, hemorrhagic, or hypertrophic, and sometimes is serous. The two primary divisions will be separately described.

CEREBRAL PACHYMENINGITIS EXTERNA.

DEFINITION. It is an inflammation of the external or periosteal layer of the dura, which is almost universally secondary to injury, or disease of contiguous structures. It may be hypertrophic, or purulent.

SYMPTOMS OF A TYPICAL CASE. A person who has been suffering from an injury of the cranium, or an inflammatory disease in scalp, face, ear, nose, or orbit, becomes comatose or has convulsions of the Jacksonian type, followed by paralysis of the monoplegic variety. This paralysis is remittent, and unaccompanied by sensory disturbances, or aphasia. The last stage may be marked by one-sided optic neuritis.

DIFFERENTIAL DIAGNOSIS. This is from Tumor, Syphilitic Meningitis, Tubercular Meningitis, Cerebral Apoplexy, Cerebral Thrombosis.

AGE. Very rare in childhood.

ETIOLOGY. A severe injury to the cranium is extremely likely to involve the external layer of the dura; obviously certain to result from fracture, or penetrating wounds of the skull. If caries, or necrosis of the bones of the skull results from disease of the middle ear, the nasal cavity, or the orbit, the dura is usually implicated. A syphilitic new growth of the skull is a common cause, and erysipelas of the face and head may excite it by way of the veins of the diploë.

PATHOLOGY. If the exciting cause is a non-infected injury, it will result in the hyperplastic form. There will be an abundant

cell-proliferation, and the membrane will become thickened, and adhere strongly to the bone, instead of being easily separable as in health. This condition generally is true of a small area only. If the exciting cause is an injury with infection, or an extension of a pre-existing infection, we find the purulent type, limited to a small area, as is true of the preceding type. In this case we find the dura separated from the cranium by a collection of pus, practically an abscess of the dura. This process may infect the whole structure of the dura, and involve the pia in a general infection, which will result in a matting together of all the membranes. In other instances the collection may become so great as to produce a bulging of the dura.

SYMPTOMATOLOGY. In general it will not be easy to separate the symptoms arising from the original disease and those additional ones due to the subsequent implication of the membrane. Usually, however, we shall find a more pronounced and constant headache, and if there is a collection of pus, the pushing inward of the membrane will practically amount to a tumor, and will exert pressure upon the brain, and may produce weakness or paralysis on the opposite side of the body. Such a projection will also interfere with the circulation in the vessels lying below it, and will disturb the functions of the brain in the areas to which these vessels are distributed. If the meningitis occurs at the site of a sinus, it will induce a primary sinus thrombosis by an extension of the inflammatory process, or by simple compression. A rise of temperature dependent upon the extension to the meninges is rare.

COURSE AND PROGNOSIS. The condition is essentially chronic, although it may be acute in the purulent type. The prognosis is serious, but less so than in the other forms of meningitis.

DIAGNOSIS. Rarely diagnosed during life. It is to be inferred from an increase in the gravity of the symptoms of the primary disease. It has a large basis of probability if to the symptoms of the initial disease are added mental disturbance, and is quite certain when the opposite half of the body gradually develops some grade of paralysis.

TREATMENT. The treatment is distinctly surgical, and may be addressed to the primary process, or, if the area is accessible, it might extend to the drainage of the purulent collection, or to the clearing out of the thrombosed sinus.

PACHYMENINGITIS INTERNA.

(*Hemorrhagica, or Hematoma of the Dura.*)

DEFINITION. This is a chronic inflammation of the inner layer of the dura, either serous, purulent, or hemorrhagic.

SYMPTOMS OF A TYPICAL CASE. No typical symptoms can be given.

DIFFERENTIAL DIAGNOSIS. This is from Tumor, Syphilitic

Meningitis, Tubercular Meningitis, Cerebral Apoplexy, Cerebral Thrombosis.

AGE. It is found in very early life, and also in late life.

SEX. It is more frequent among males than females.

ETIOLOGY. The serous variety is very rare, but is found in young children, in dementia paralytica, and in chronic hereditary chorea; i.e., in diseases diminishing the bulk of the brain. It is called external hydrocephalus. The purulent variety is also rare, and may be the primary result of infection, but is more likely to be the result of an extension from the external layer of the dura, or from the pia. The hemorrhagic variety vastly outnumbers the others, and is the result of an accidental rupture of a meningeal artery which has been weakened by the changes incident to old age, phthisis, cardiac, and renal disease, the exanthemata, erysipelas, and cachectic and purpuric states. It principally occurs in the course of chronic alcoholism and dementias, especially in the paretic type.

PATHOLOGY. The serous form results from an exudation from the vessels, and probably minute capillary hemorrhages, which result in an extremely thin membrane over the inner surface of the dura, and an increase in the free fluid in this locality, which is normally only sufficient to moisten the surface. The purulent form shows adhesions of the membranes, and greater or less accumulations of pus. The hemorrhagic variety shows a condition which is the result of hemorrhage and hyperplasia. It is not as yet decided whether a toxin has first caused a proliferation of the epithelial layer of the dura into a tremendously increased bulk which is traversed by frequent blood-channels which break down and leave hemorrhagic spots, or whether the vessels first break down, and the layers of fibrin are the result. However initiated, the result is a fibrinous deposit, layer on layer, sometimes as many as twenty, which being full of blood-channels with poorly organized walls, is the seat of repeated hemorrhages. The new hemorrhages, as they are called, separate the older layers, or form new ones on the surface toward the brain. A mild case will produce a thin, pinkish deposit on the dura which may easily be peeled off, but typically there is a tenacious mass, with perhaps twenty layers, marked with the yellow and brown discoloration of recent and old hemorrhages. There may be a mass as large as a goose egg. As a natural result, the bones of the skull may be thinned out by the pressure of the new growth, or on the other hand the cerebral convolutions may be flattened. In children, this increase of cranial pressure will retard the closure of the fontanelles, and the ossification of the sutures. This condition is usually found in a circumscribed patch at the vertex, in the distribution of the middle meningeal over the motor zone, but occasionally in the basal fossae. It may extend over one or both hemispheres.

SYMPTOMATOLOGY. Symptoms may be absent, and are generally indefinite. This pathological condition, however, has been found post-mortem in cases presenting now some, now others, of the following symptoms: A prodromal period of excitement with physical restlessness. This is followed by a severe headache, which is in turn followed by a coma, which may persist with a retarded pulse for days and weeks. This coma may be intermittent, a peculiarity which marks many of the symptoms. Instead of coma there may be apoplectiform attacks, focal or Jacksonian epilepsy, or localized spasm, or, less commonly, convulsions. The headache may be limited to the vertex, or temporal region at the onset, and it may be accompanied by vomiting. In the coma, paralytic symptoms are apt to develop, and may be monoparesis, or of one side afterwards spreading to the other. This paresis may be accompanied by rigidities and contractures, or may be free from them. The paralysis is remittent. There are awkwardness in movement, loss of memory, and of intellectual capacity. Sensation is rarely affected, nor is aphasia often present. Conjugate deviation of the eyes has been observed, and also nystagmus, while optic neuritis, and choked disc, often of the opposite side, are liable to appear in the final stages.

COURSE. It is a chronic disease and since the early symptoms are not diagnostic, we have no data from which to detect the date of its origin. For a long time there will be indefinite symptoms of illness, but later there will be apoplectiform seizures, or comatose conditions for a limited time, which will always leave certain vestiges behind in the shape of weakened intellect, or some degree of paralysis.

PROGNOSIS. Rarely cases have terminated in recovery, with an absorption of the exudate, but usually there is an incurable primary condition, of which this is a secondary result, and therefore the ordinary conclusion is death.

DIAGNOSIS. Rarely diagnosed during life. If, however, a case of chronic alcoholism, or general paresis develops a chronic weakness of the limbs, and mental failure with more or less of the symptomatology detailed above, we should be justified in hazarding a diagnosis.

DIFFERENTIAL DIAGNOSIS. Tumor. This subdural thickening practically amounts to a tumor, and no distinction can be made, unless the etiological basis for pachymeningitis interna is very evident.

Meningitis of any type is difficult to distinguish while the patient is comatose.

Syphilitic Meningitis is to be diagnosed from the prominence of the nocturnal headache, and symptoms of specific disease in other parts of the body, and a history of exposure, or habits which would suggest its great probability.

Tubercular Meningitis in Children. This differs from pachymeningitis interna in the presence of constipation, retraction of the abdomen, severe headache, rigidity of the neck, and marked troubles with respiration and circulation. The cranial nerves at the base, except the optic, are spared in pachymeningitis.

Cerebral Apoplexy. The possibility of confusion would exist only in the case of adults, and even then, the rapidity of the onset, and the greater permanence of the results would give us grounds for differentiation. In pachymeningitis there are intercurrent convulsions, and frequently choked disc.

Cerebral Thrombosis. This may easily be confused, since the symptoms in their gradual onset, and transitory nature, are quite similar.

TREATMENT. This is identical with that for the inflammation of the same membranes in the spinal canal, and therefore will be found at the end of that article.

PACHYMENINGITIS SPINALIS.

MENINGITIS affecting the spinal dura is divided into external and internal, as in the cranium, but, since the spinal dura is not directly applied to the vertebrae as the cerebral dura is to the skull, the condition arises from different causes, and leads to different results. When the internal layer is the seat of inflammation, the other membranes are as commonly implicated as when the disease is located in the cranium, but, since the septa of the cord are merely prolongations of the pia, the secondary damage to the cord substance finds no counterpart in the case of the similar disease in the brain.

PACHYMENINGITIS SPINALIS EXTERNA.

(External Pachymeningitis. Peripachymeningitis and Perimeningitis.)

DEFINITION. This is a form of inflammation of the dura which is not confined to the external layer of the dura, but involves also the fatty tissues, and the veins which lie between it and the vertebrae. It does not tend to implicate the inner layer of the dura, even when the inflammation is most intense. Under some conditions all the membranes are involved in a common inflammation, and the cord is constricted by the new-growth. The inflammation is of the chronic type.

SYMPTOMS OF A TYPICAL CASE. After a preliminary disease in some part of the spinal column, the pain in the back increases, and there is a change of sensation in the arms. This is succeeded by

twitchings in the muscles, then loss of power and sensation, followed by atrophy with the reaction of degeneration, and by contractures. Later there may be a spastic paralysis of the legs, and as a final stage this paralysis may become flaccid with disturbance of the sphincters. All these symptoms are developed slowly and intermittently.

DIFFERENTIAL DIAGNOSIS. This is from Multiple Neuritis and Myelitis.

ETIOLOGY. Injuries to the spinal column may cause it, and it has been considered to have resulted from the infection of purulent foci in the immediate neighborhood. The present belief, however, is that it practically always results from osteomyelitis, and that when near-by foci of pus have been found they are extensions from the spinal canal, and not the opposite. Syphilitic new-growths and the extensions of cancerous infiltration are occasional causes.

PATHOLOGY. Examination reveals a hyperplastic thickening of the dura to all degrees, from a slight increase, to a sort of nodulated tumor, which may invade the subdural space, or fill it solidly to the vertebrae, or even necrose these bones by its pressure. The nodules may be scattered about, or be aggregated into one mass. The changes are commonly limited to the immediate vicinity of the diseased vertebrae, but in some instances have been found to extend throughout the whole length of the canal. The fat in this space disappears, and the overgrowth often unites the dura and the periosteum of the spinal canal. The surface of the dura is sometimes only reddened with a little lymph upon its surface, but usually the deposit is purulent, and the amount may be so large that it invades the surrounding structures. It may be sufficient in quantity to compress the cord.

SYMPTOMATOLOGY. It will render the symptomatology more evident if we observe that it mainly arises from the compression and inflammation of the nerve-roots. This is a necessary result, since they pass out of the spinal canal through the pia and the dura, and their coverings are prolongations of these membranes. There is tenderness over the spine, at the vicinity of the inflammation, and shooting or constant pain of quite an intense character in the distribution of the implicated nerves. The pain in the back is sometimes referred to the region of the loins. The skin in the distribution of these nerves is hyperesthetic. The muscles become rigid, and movements of the back are usually restricted and painful. They are the seat of twitchings and spasm. As the compression of the nerves becomes more intense, anesthesia takes the place of hyperesthesia, and muscular twitchings are replaced by paralysis. This is an evidence that degeneration of the nerve-fibres has been produced. When the secretion of pus has been sufficient to compress the cord, we shall have a compression myel-

itis with a spastic paralysis, and hyperesthesias followed by flaccid paralysis and anesthetics, paralysis of the sphincters, lividity of the skin, and perhaps bedsores. In such acute cases there will be rigors, and fever up to 110° , and profuse sweating. The completeness, or incompleteness, of such results will indicate the degree of compression.

COURSE AND PROGNOSIS. In some cases it runs an almost unnoticed course, and continues for a long time. The primary affection usually determines its outcome, and when it results from caries, with a cure of this condition, there may be a return to health after a long-continued compression of the spine, if it has been moderate in degree.

DIAGNOSIS. The causative condition will commonly give rise to the symptoms which would direct one's attention to the diseased locality. Allowing the existence of the symptoms, we should judge that an external pachymeningitis had resulted, if paresthesias, and muscular twitchings, and a wider distribution of the spinal pains were discovered. If the spinal muscles showed a deep edema, we should suspect a purulent meningitis; and collections of pus in the pleura, posterior mediastinum, back of the abdomen, or behind the pharynx, or sudden compression of the cord, would indicate it all the more forcibly.

DIFFERENTIAL DIAGNOSIS. Multiple Neuritis differs in these points. In this disease all four limbs are usually involved, while in pachymeningitis the legs are usually free. The anesthetics of multiple neuritis are on the distal portions of the limbs only, and the trunk is free, while the contrary is the case in pachymeningitis. There is no tenderness of the nerve-trunks in pachymeningitis, and there is evidence of injury to the cord in some cases.

Myelitis has an acute, and not a chronic onset, no pain except a girdle sensation, has early paralysis, bladder and bowel symptoms. Pachymeningitis runs a chronic course, has more pain, spasm and irritation, and the paralysis is a late symptom.

TREATMENT. This is covered primarily when the osteomyelitis has been corrected by the application of orthopedic apparatus. Calcareo carb., Fluoric and Nitric acid, Silicea, and Hepar sulph., in the lower potencies, or in the form of Calcium sulphide, will now have a decided influence upon the necrotic process in the bones and the absorption of the resulting exudates. If the cause is syphilitic, the treatment should be specific, and if carcinomatous the case is a hopeless one.

PACHYMEINGITIS SPINALIS INTERNA.

DEFINITION. This is an inflammation of the chronic type in the internal layer of the spinal dura. While it may be extensive in its distribution, the majority of the cases are confined to the cer-

vical region, and this variety is termed pachymeningitis cervicalis hypertrophica. It is probably a hemorrhagic condition, and while it generally involves the soft membranes in the inflammation, and also the substance of the cord, it, so far as symptoms result, is a proliferation and a fibrinous thickening of this membrane, with the production of symptoms depending upon the compression of the nerve roots.

THE SYMPTOMS OF A TYPICAL CASE. After a period of indefinite pains in the spine, and stiffness of the neck and back, there is a disturbance of sensation in the forearms, followed by muscular twitchings, then loss of sensation, and finally paralysis of the arms. This paralysis is followed by contracture, in either over-extension or flexion. If the lesion is in the lumbar region, there is the same condition in the legs with disturbance of the sphincters.

DIFFERENTIAL DIAGNOSIS. This is from Syringomyelia, Diseases of the Spine, Progressive Muscular Atrophy, Transverse Myelitis, Tumor, and External Pachymeningitis.

AGE. Adults are usually the victims, but sometimes it affects children.

SEX. Males almost always, from causes disclosed by the usual etiology.

ETIOLOGY. Syphilis, alcoholism, traumatism, and exposure, but the two former are the almost certain causes.

PATHOLOGY. Wherever it is located, the primary process is the rupture of an artery, or a fibrinous exudate from it. The primary layers are therefore of fibrin mixed with blood, while the later ones are more purely fibrinous. The character of the process determines that the layers should be reddish brown at the base, with clearer ones superimposed, which are spotted with the stains of hemorrhages. This fibrinous deposit shows all degrees of hardness up to ossification. When the other membranes become involved in the process the circulation of the cord is compromised, a marginal myelitis is set up, and as the pia sends septa into the substance of the cord, between the layers of which develop the lymph channels of that structure, there follows a lymphatic congestion in the cord which produces cavities, and the general process may induce a myelitis. Connective tissue may extend along the blood-vessels, and the compression of these vessels will produce areas of softening. Gliomatous overgrowth may also be a result. When the process is purely of the dura the emergent nerve-trunks are compressed, and they may become atrophied and sclerosed. A compression myelitis may be set up by the pressure of the exudate developed upon this inner layer of the dura. If the soft membranes are also affected, the first result will be a marginal myelitis, while if the substance of the cord undergoes a pathological change there will be any grade of myelitis, up to a complete transverse section. The distribution of this

process is sometimes very extensive, and may extend the whole length of the cord; it generally is confined to a small vertical area in the cervical region, and to the upper, or the lower part of the cervical enlargement. When the cervical region is affected there may be an extension to the medulla and pons, or, on the other hand, the process may be just as closely confined to the lumbar enlargement. The cerebral dura is often affected at the same time.

SYMPTOMATOLOGY. The disease is essentially chronic, and has been divided into three stages, which may be separated by years and months. These are the neuralgic, the paralytic, confined to the arms, and the stage where there are spastic symptoms in the legs. In the first stage there will be pain in the neck, and perhaps in the back of the head, and between the shoulders, and the spinal vertebrae will be tender to percussion. There will be tension and stiffness in the neck, and the head will be carried forward, and motion to either side will be restricted. The pains in the arms will be of an intense neuralgic type, especially along the ulnar and median nerves, and there will be slight muscular stiffness in the arms and hands. These symptoms depend upon the compression of the posterior nerve-roots, and generally last from two to five months; but it may be years before the paralytic stage sets in. This is caused by degeneration of these nerves. The pains continue, but now areas of anesthesia appear upon the members. The muscles affected will be those innervated by the median and ulnar, if the lower part of the cervical enlargement is involved, and the musculo-spiral group, if the disease is in the upper part. The musculo-spiral is most usually the seat of the lesion, and therefore the flexors are most generally impaired. Many authors have taught that there is a characteristic deformity, the so-called "Preacher's Hand," in which there is over-extension of the wrist joint, extension of the basal, and flexion of the middle and terminal phalanges, but while common, this is not universal, nor diagnostic, depending for its presence upon the site of the lesion. It is, however, a fact that the small muscles of the hand most universally suffer, and that both arms are together, but not equally, affected. The affected muscles atrophy, and display the reaction of degeneration. If the bulb or medulla shares in the changes, there may be a bilateral impairment of hearing, and tachycardia, or the whole symptom complex of bulbar palsy. Oculo-pupillary symptoms sometimes occur, but without any diagnostic peculiarity. If the lesion is confined to the lumbar region there will be pain in the distribution of the nerves of that locality, rigidity of the muscles, and finally talipes. In the third stage the cord shows the signs of compression, and there will be a spastic paraplegia of the lower limbs, with increased reflexes, and disturbance of the sphincters. If a myelitis has been set up, the symptoms will be such as are found described under that caption.

COURSE AND PROGNOSIS. The disease is very slow in onset, and in passing to its successive stages. Patients have been known to survive for twenty years. The prognosis is usually grave, unless it is diagnosed at an early stage. It has been known to stop at any stage, and recoveries are by no means unknown. While pain tends to decrease with the advent of the paralysis, patients have been known to succumb to the distress of the pain and the rigidities.

DIAGNOSIS. The diagnosis of a spinal meningitis of the internal layer of the dura is to be made from the appearance of symptoms of a primary compression of the nerve-roots, especially in the cervical region, without a pre-existing spinal disease to explain the condition. If it is in the later stages the true origin of the process would be suspected from the fact that the process was a very chronic one, and that the symptoms of nerve-irritation had antedated the paralysis by months or years. When all the membranes are affected, or the cranial dura is also affected, the diagnosis may be impossible. The cerebral symptoms may overshadow the spinal.

DIFFERENTIAL DIAGNOSIS. Syringomyelia. This very closely resembles pachymeningitis cervicalis in its general character, and distribution, since in each case there are muscular atrophy in the arms, and paralysis and wasting in the legs, but in syringomyelia the pains in the arms are usually much slighter than result from pachymeningitis, and there is rarely the early limited impairment of all forms of sense-perception which results from damage to the nerve-roots in pachymeningitis, and syringomyelia is a far more chronic disease.

Diseases of the Spine. These are often capable of exclusion from the history. In addition to this the root-pains are rarely very severe, and tenderness and irregularity of the vertebral spine can be discovered by examination soon after the commencement of the disease. This practically differentiates external pachymeningitis also.

Progressive Muscular Atrophy is free from the severer degrees of pain, and is often without any sensory symptoms, and has none of the stiffness and rigidity so conspicuous in the disease in question.

Myelitis has segmental symptoms before the neuritic ones. It is usually much more acute, and in any case the symptoms of paralysis, and disturbance of the sphincters precede, and do not follow, the pains and changes of sensation. If the invasion has been rapid and widespread, it is impossible to differentiate cervical myelitis from pachymeningitis cervicalis hypertrophica.

Tumor. If it involves the nerve roots it cannot be diagnosed from a pachymeningitis, since its results are the same, but generally speaking the symptoms of tumor are compression of the

cord, rather than irritation of the nerve roots, and it differs in two other peculiarities: first, that the vertical area is considerably greater; and secondly, that the effects are usually bilateral from the first.

TREATMENT. The posture of a patient with internal pachymeningitis should be such as to drain the spine, and it has been suggested that one of the best methods is to keep the patient as much as possible upon the side or face, and this may be accomplished by arranging a padded board, or a bolster, or other form of bed-rest, against which he can lean. Warm baths are of value, and hot packs and sweat baths have produced good results. Galvanism to the spine should always be employed in such a manner as to include the involved area between the poles. Counter-irritation by Iodine, or the actual cautery, benefits some cases. Operation has been suggested, and should be thought of as a possibility. The danger is, however, far more than that involved in gaining access to the spinal cord, and rests largely upon the probability that the removal of the diseased tissues will cause irreparable injury to the cord.

THERAPEUTICS. Remedies to be used for the condition are the following: Aconite has been advised in the preliminary stages where we get signs of inflammation, and tearing pains in the nape of the neck, with stiffness and pain running to the shoulder, particularly the right. This idea of its adaptability rests upon the similarity of the peripheral pains and numbnesses to those of the drug. These, however, are the results of mechanical compression of the spinal nerves, and only come after the exudation on the meninges has become very pronounced. The congestive stage without exudation, which is the proper field for the use of Aconite has passed long before the condition is susceptible of diagnosis. Bryonia is always to be thought of in the stage where we find an exudative inflammation of the serous membrane, and the presence of the pain is no bar to the use of the drug. It is probably our most efficient remedy. Cuprum acet. is specifically a spinal cord remedy, with clonic spasms which begin at the extremities, and there is a paralysis of all the muscles of the back up to the neck. There is much nausea, vomiting, and torpidity of the bowels. Ferrum phos. is quoted by Bartlett as a remedy for the early febrile stage, with a soft and full pulse, before exudation has taken place. Hepar sulph. should be thought of when there is suppuration. Oxalic acid when the cord has become affected, and there is a meningo-myelitis as a result. Iodoformum is suggested by O'Connor on account of its favorable action in cerebral cases. Silicea in suppurative conditions generally. Secale cornutum for tenderness of the lower cervical and upper dorsal vertebrae, with stiffness of the neck, tingling of the back which extends to the fingers and toes, and pressure upon the parts affected causes pain

there and through the chest, which is aggravated by every exertion or strain upon the spine. Agaricus for the cases where the spinal inflammation is combined with that of the cerebral meninges. With the ordinary cerebral symptoms there are drawing pains in the occiput, painful sensitiveness of the scalp, stiffness of the nape of the neck and back, violent pains along the spine, with stiffness and soreness, and liability to convulsions. *Cicuta vir.* has the same combination of cerebral and spinal symptoms, with a great stiffness of the neck, and tension and soreness of the muscles with a retraction of the head. There are numbness and distortion of the limbs, and a marked concomitant is gastralgia with vomiting.

Physiological remedies are the Bichloride of Mercury, or Mercury by inunction, and the Iodides, largely used in the belief that they are absorbents of new growths and exudates, and also in view of the possible syphilitic origin of many of the cases. The other remedies are sedatives in some form, as no other definite medication has thus far met with acceptance.

LEPTOMENINGITIS SPINALIS.

This rarely occurs, except as a complication of the form of pachymeningitis just described, or as an extension of the cerebral type, and is sufficiently described under those heads.

ACUTE CEREBRAL LEPTOMENINGITIS.

DEFINITION. This is an acute inflammation of the Pia-arachnoid covering the brain, and in about one third of the cases it extends into the meninges of the cord. The meninges are not always affected as a whole, but in such definite areas that we are forced to recognize a more or less definite localization upon the Convexity, at the Base, and in the Ventricles, as well as a diffuse form which is called Cerebro-spinal. There is a diversity also in the kind of process affecting the tissues, and therefore it is divided again into Purulent, Serous, Tubercular, and that following a rather definite group of causes of infection, which is called Epidemic Cerebro-spinal, and also that from Syphilis.

THE SYMPTOMS OF A TYPICAL CASE. Except in tuberculous cases there is an initial fever, with an initial delirium, with the speedy addition of a severe headache which lasts through all stages of the disease. It is marked by vomiting, and a rapid prostration of the mental and bodily powers. There is constipation, but urination is involuntary. Choked disc rapidly comes on, and there is failure of vision, and of hearing, and the cranial nerves are quickly affected, giving rise particularly to strabismus. The irritation of the cortex is shown by Kernig's sign, and the vasomotor paralysis by the "tache cerebrale." Irritation of the

spinal nerves produces a stiff neck, and an antero-posterior curvature of the spine.

DIFFERENTIAL DIAGNOSIS from Typhoid Fever, Febriculae of Children, Rheumatism, Muscular (nuchal) Rigidity from other causes, Intra-cranial Tumor, Hydrocephaloid of Marshall Hall, Hysteria, Meningeal Hemorrhage.

AGE. While it may occur at any age, the greatest number of infantile cases occur between the third and sixth month (Barlow's statistics from Starr), and the majority of all such cases occur during the first and second years of life. Children, more often than adults, suffer from the Cerebro-spinal form. Septic occurs at any age. Tuberculous from two to fifteen years most frequently.

RACE AND HEREDITY are not factors.

ETIOLOGY. Osler (Cavendish Lectures, 1899) classifies the causes as follows. They are first divided into Primary and Secondary:

PRIMARY.

- (1) Cerebro-spinal Fever.
Which is either Sporadic or Epidemic. Caused by the *Diplococcus intracellularis*.
- (2) Pneumococcic.
Meninges alone involved, or share in a general pneumococcus infection. Caused by *Pneumococcus*.

SECONDARY.

- (1) Tuberculous.
Caused by *Bacillus Tuberculosis*.
- (2) Pneumococcic.
(a) Secondary to Pneumonia, Endocarditis, etc.
(b) Secondary to injury of the cranium or its fossae, or to disease. All caused by the *Pneumococcus*.
- (3) Pyogenic.
(a) Following local disease of the Cranium or a local infection elsewhere.
(b) Terminal infection in various chronic maladies. Both *a* and *b* are caused by various forms of *Staphylococci* and *Streptococci*.
- (4) Miscellaneous acute infection.
In Typhoid Fever, Influenza, Diphtheria, Gonorrhea, Anthrax, Actinomycosis, and other acute diseases.
Caused by the Typhoid bacillus, Influenza bacillus, Diphtheria bacillus, *Gonococcus*, etc.

PATHOLOGY. Infection of any form can reach the meninges, either from injuries which penetrate the cranial vault and lay

them open to external causes of infection, or from near-by foci of purulent affections of the ears, nose, frontal sinuses, etc. The most common source of such infection is purulent disease of the middle ear, or of the cells of the mastoid, which conditions are common results of influenza. The other source of infection is from micro-organisms in the general circulation. The primary effect of such infection is to thicken the coats of the vessels, and infiltrate them by proliferation of round cells derived from the fixed connective tissue, and by a migration of leucocytes. The vessels may finally rupture, but, in any event, the meninges are thickened by this exudate, the lymphatics are plugged with debris, and, moreover, the exudation from the surface in certain areas binds the arachnoid and pia and dura together, which should be free to move upon one another without resistance, since their function is simply to support without impediment the blood and lymph channels supplying the brain. In purulent cases the exudate becomes a yellowish green and sticky fluid, which is most abundant along the sulci, but more or less over the whole surface of the meninges. It insinuates itself along the sheaths of the cranial, and upper spinal nerves, and thus inflames them. Owing to the rupture of arteries, and plugging up or obliteration of veins and lymphatics, the superficial layers of the cortex are the site of serous infiltrations, small hemorrhages and foci of pus. While a purulent meningitis confined to the ventricles is said to occur sometimes in childhood, such an invasion is, to some degree, a feature of all cases. It is due to a congestion of the choroid plexus, or an inflammation of the ependyma, from which results an exudate similar to that upon the cortex, by which the foramina of the ventricles are occluded, and a condition of hydrocephalus results. The cranial nerves are not all equally affected. The third nerve is especially liable to invasion, and therefore strabismus is a most constant symptom. The paralysis of the facial and optic are next in frequency, while the olfactory almost always escapes. This purulent exudation is lacking in the meningitis of infants, and also in the tuberculous form, but is replaced by a serous exudate composed of serum and lymph, which appear as flocculi in the thickened cerebro-spinal fluid.

SYMPTOMATOLOGY. The symptoms of the various varieties of inflammation of the pia-arachnoid have so many general symptoms in common, that it seems best to describe the condition as a whole, modifying it from time to time in points where specially marked variations occur. It will, however, not be amiss to review briefly a few facts showing how the localization is affected by the etiology, and, flowing from that, how the symptomatology is, to a certain extent, dependent upon broad differences in localization. For instance: the epidemic form affects the entire pia, both cerebral and spinal, while if it is a secondary disease, depend-

ent either upon injury, or foci of infection within its immediate vicinity, we shall find a septic form which has a peculiar predilection for the meninges of the convexity, and perhaps only a small portion of this, even; the base may entirely escape. The primary meningitis of infants attacks especially the posterior basal region, while the tuberculous form attacks the base as a whole, from whence arises the frequent synonym for this form: namely, "basilar meningitis." If syphilis is the primary cause of the meningitis, we shall most certainly find the seat of its sole, or greatest activity at the base, near the posterior perforated space, and spreading thence over the crura cerebri, and the sides of the medulla and pons.

What variation of symptoms should we reasonably expect from these variations in localization? If the convexity is affected, delirium, local convulsions, and hemiplegic weakness are common, vomiting is somewhat rare, palsies of the cranial nerves and optic neuritis, for the most part, are lacking. Localization within a limited area of the convexity would be indicated by monoplegia. If the meninges at the base of the brain are the seat of inflammation, the cranial nerves suffer early, vomiting is frequent, optic neuritis usually develops before the symptoms have reached a high degree of intensity, and delirium comes later. We may draw the conclusion that not the whole, but only a part of the basal meninges are implicated, when we find the cranial nerves affected upon one side only. When retraction of the head is an early and strongly marked symptom, it implies that the membranes about the pons and the medulla are affected. No one of the above localizations excludes the possibility of ventricular damage, since, if the disease is of the convexity, the inflammation may extend into the ventricles by following the infolding of the pia, while if of the base, the foramina of communication between the ventricular and the cerebro-spinal space furnish an avenue by which infection may enter the ventricular cavities, and in addition produce indirectly a hydrocephalus by an adhesive inflammation about these openings. A primary meningitis of the ventricles is said to be possible, but its symptoms are not distinctive, except that the cranial nerves are not generally affected. All cases of cerebral leptomeningitis, whatever be the cause or localization, will present, to some degree, the following conditions and symptoms.

Headache. It is generally an initial symptom, although some purulent cases, and those to some degree of a serous character from altered states of the blood, may not exhibit it. It is generally frontal, or occipital, but may be diffuse. It is peculiarly unmanageable, and lasts throughout the disease with such intensity, that while the patient is conscious he complains of it bitterly, and, even in coma, the face may be seen to scowl with

the suffering. Its intensity gives rise to the shrieks which are known under the name of the "Hydro-cephalic Cry," and causes the patient to rub the head upon the pillows, or continually grasp it with the hands.

Vertigo. This occurs at times, and persists so long as the patient is conscious, but it is often obscured by other symptoms, so that it is not noticeable.

Delirium. This comes on in a short time, varying from a few hours to as many days, according to the severity of the process. The patient shows from the first the effect of the disease upon the sensorium by being deficient in the power of attention, and his replies are short and irrelevant. It is very variable in degree, and is usually low in grade, but, from the mixture of pain and intoxication, it may be wild, or busy, like that of typhoid or alcoholism. As a diagnostic point, its value rests upon its association with headache. At first it may be only at night, or in sleep, but, once developing, it is persistent.

Vomiting. Cerebral in type, i.e., projectile, without previous nausea, and at first with a clean tongue, which later becomes foul, and with a bad breath. The stomach is simply intolerant of food. It may be a very early symptom.

Temperature. The type of disease is no more diverse than the degree of the rise in the temperature, and this will be specifically stated later. In the typical form, however, while a rare case develops no fever, it will steadily rise until it attains a height of 104° in a few days, and toward the end of a fatal case it will go to 108° , or, on the contrary, become subnormal. Even in cases where the temperature is high it does not always show an evening rise, nor is the final elevation regular in its development, but fickleness is its characteristic. These irregular variations are ascribed to the development of some nervous conditions which neutralize the tendency of the inflammatory process to reveal itself by an increase in the body-heat.

Chills. Almost always a feature of the onset, and when absent, supposably, may be slight, or unobserved. They may recur in the course of the disease.

Pulse. Rulably increased, but diagnostically erratic so far as following the ordinary rules of the relation which should obtain between pulse and temperature. In the early stage, which is one of excitement, it is usually full, active, and greatly accelerated, but, even at first, it may be 50 or lower. Toward the termination of a fatal case it may still be low, but usually is high, and may be uncountable. It is liable to great variation within very short periods of time.

Respiration. This is not so much disturbed as one would expect, and, like the pulse, does not follow the temperature. Generally it is quickened in the early stage, and becomes slow

and irregular with the advent of coma. Toward the final stage, it is often Cheyne-Stokes in character, although it is a feature of the earlier stages of the Tuberculous form, so that while in most cases it is a sign of death, such is not always the fact. There is another peculiar condition in the course of the disease. The patient is prone to periods where the respiration ceases for several seconds at a time, and, in meningitis of the posterior fossa, there will suddenly occur a failure of breathing with irregular periodicity, cyanosis, and death.

Nuchal Rigidity. From the first a large proportion of cases will complain of audibly, or by their actions reveal, a rigidity of the muscles of the neck. The patient will attempt to get the head back over the pillow, or in the early period of more gradual onset drop the head over the back of a chair, or resist attempts to passively draw the head over toward the chest. It is one of the first unequivocal symptoms, and as the inflammation progresses down the spinal column, it may extend to the muscles of the back in general. It arises from the irritation of the spinal nerve-roots, and is an indication for the determination of the lowest level of the meningitis.

Muscular Rigidity in General. This rigidity is not confined to the neck and back alone, but the board-like rigidity, and even retraction, of the abdominal walls is from the same cause in some instances, and in others is a symptom of irritation of the motor cortex of the brain. Tapping the muscles causes the patient to shrink, and percussion of the spinal muscles causes a retraction of the spinal-column. The muscles of the limbs are in a state of irritability, and this is the cause of what is known as Kernig's sign. When this sign is present it is impossible to fully extend the leg so that it is in a straight line with the thigh, if that has been previously placed at a right angle with the trunk.

Test. Place the patient on the edge of the bed, or in a chair, or lying in the bed upon the back. If on the edge of the bed, or in a chair, the thigh will be at right angles with the body, but if in the bed, it must be so placed. Now, with the thigh in this position, try to bring the lower leg in a line with it, and it will be found to become fixed at an angle of less than 160 degrees. The arm will at times show the same phenomenon. This is a sign almost universally present, and when found is conclusive evidence that there is irritation of the meninges. At times trismus will be present, but it is a rare symptom, although this rigidity is more common than a paralysis of the muscles of mastication.

Hyperesthesia. One of the earliest signs is a hyperesthesia of the Special senses. This is shown by the desire of patients to be left in a darkened room, and their evident annoyance at the least noise about them. The same is true of the muscles, and is

referred to under the head of Muscular Rigidity. The skin is sensitive to pressure, and the tissues of the skull also, since the patient will wince and shrink from percussion. A unilateral hyperesthesia is rare, and hemianesthesia is unknown.

Vasomotor Disturbance. This is an early sign in all cases, and under its influence the skin assumes a dark and sometimes mottled appearance. If the finger-nail be drawn quickly across the skin it will be followed by a dark-red discoloration. This is the "Tache Cerebrale," but it is not diagnostic of meningitis, as such a degree of vasomotor disturbance is seen in many other diseases.

Deep Reflexes. These are usually increased at the outset, but tend to be decreased later in the disease. If the patient survives for a week or ten days, they are generally obliterated. There may be a discrepancy upon the two sides of the body. In some cases, there will be sufficient descending degeneration of the motor-tracts, to develop Babinski's toe-sign—extension of the great toe upon stroking the sole of the foot.

Bowels. These are almost universally constipated.

Bladder. The urine is retained as stupor comes on, but occasionally there is incontinence instead. The quantity is diminished, as we should expect in the febrile condition, and it is often albuminous, but very rarely does it contain sugar. If the floor of the fourth ventricle is irritated, we may find both albumin and sugar in large amounts.

Cortical Irritation and Softening. Convulsions are common, especially in children, and are usually general and protracted, but they may be upon one side only, or of a single part. The tendency is toward generalization, irrespective of the site of the lesion, since irritation is not so sharply defined, and the whole brain is in a state of excitement. These may occur at any stage, although very common as an initial symptom in severe cases, and may be the only symptom present in septic ones. Beside convulsions, we find twitchings of the muscles, and paralyses of a side, or of a single part; and facial palsy of a cerebral type, i.e., leaving the upper face free, may be a symptom at any stage. Aphasia is common in the Tubercular forms, but rare in the others.

Asthenia. A general muscular weakness is always present, and may be quite sharply localized in an intense form in those cases where the process is definitely localized.

Cranial-nerve Involvement. Few cases spare the cranial-nerves entirely, but the facial, the oculo-motor, and the optic (the olfactory far less often), are involved to some degree in almost every case. The exudate burrows along the sheaths of the optic and the facial nerves more readily than it is possible in the lower cranial nerves, and, since the auditory and facial are in close connection in the bony canal of the external auditory meatus, this

latter nerve is also frequently attacked. By this method of attack, and not by infection of the nerves in continuity, are to be explained the cranial-nerve symptoms. The nearness of the two abducent nerves to one another at their point of exit from the pons explains the occurrence of paralysis of both external rectus muscles in basal meningitis. The early symptoms are those of irritation; we find haziness of sight, intolerance of light, and also of noise, and occasionally over-sensitiveness to odors. In a later stage these are replaced by symptoms of destruction or serious impairment of these nerves.

Optic Neuritis. This is a notable, but not a constant symptom, rarely complete enough to greatly injure the sight, and common in cases affecting the base, while it is rare in those of the convexity.

Pupillary Changes. They occur early, and at first the condition is one of contraction, which shows this peculiarity, viz., that one pupil will be contracted more than the other on one day, and the other will be the seat of greater contraction upon another day. At a later period the preliminary contraction is replaced by relaxation, and the pupil is dilated, and the reflex contraction to the stimulus of light is greatly reduced, or altogether wanting. The reaction to cutaneous stimulation (pinching the skin over the fourth or lowest cervical sympathetic ganglion) may be present.

Deafness. This may come on in the course of the disease from an extension of the degeneration, or inflammation to the labyrinth; it is very rarely due to the destruction of the auditory nerve-trunk.

Facial Nerve. The paralysis of this nerve is common, and is shown by change in the line of the mouth, or defect in the power of movement. It is peripheral in type when the meningitis affects the base, i.e., involves the whole face, but if the disease is of the convexity only, it will be of the cerebral type, i.e., leaving the upper part of the face in a normal condition, although it may weaken it at the outset.

Oculo-motor or Third Nerve. This is very frequently affected in some of its functions, so that the subjective symptoms of diplopia, and its objective and causative symptom, squint, are very common. It is practically always present, and if latent may be developed by having the patient turn the eyes in first one, and then in the other direction, when we shall discover a lack of parallelism between the two eyes. When visible, it may be inconstant, or present only on a particular movement. In an early stage, it depends upon the irritable overaction of one muscle or another, but in a later stage it is the result of a paralysis of a muscle.

Nystagmus is less frequent. The structures of the eye may suffer, and we shall find all grades of conjunctivitis, sometimes

choroiditis, and once in a while panophthalmitis with a destruction of the eyeball.

The Pneumogastric Nerve is often affected, and such injury is no doubt a frequent cause of the disturbance of respiration and heart's action.

The Hypoglossal Nerve is not always immune, as evidenced by the occasional deviation of the tongue.

Eruptions. Herpes labialis is as common in Epidemic Cerebro-spinal Meningitis as in pneumonia, and fully as diagnostic of the variety of the meningitis in question. In all varieties urticaria is often present; far less frequently, a red macular eruption which, when present, has given the name of "Spotted Fever" to the disease. It is more like a roseola than the petechiae of typhoid fever, but its distribution is similarly upon the trunk and abdomen.

Trophic Changes. The nutrition of the skin is at such a low ebb that mild irritants are apt to produce extensive and severe blistering, and even the heat of a hot-water bottle is apt to cause a slough. There is a strong tendency to the development of bedsores, particularly in the last stages, but it is more characteristic of the subacute than of the acute cases.

COMPLICATIONS. Sinus thrombosis and cerebral abscess are the most frequent complications.

COURSE AND PROGNOSIS. There are three fairly well-defined stages: invasion, compression, and coma. While a case may develop coma as a primary symptom, or some amount of paralysis, the usual invasion symptoms are those of excitement, showing that the cerebral cortex is in an irritated condition. Such an irritation will give rise to delirium, on the one side, and convulsions and muscular contractions on the other. This stage typically lasts about a week, and during this time vomiting, delirium, vertigo, high temperature, muscular rigidities, diffuse headache, and hyperesthesias are the prominent symptoms. The stage of compression also lasts about a week, and here function of all kinds is reduced; the head is retracted, rigidity becomes more marked, and more constant, we find squinting, and other paralyses, incontinence of the sphincters, and a very slight response to the strongest stimuli, since apathy is increasing toward the last stage, which is coma. In this second stage, of compression, irregularity of respiration is very marked, either from the compression directly, or from involvement of the pneumogastric nuclei in the disease process. The last stage, or that of coma, presents deep stupor, loss of the deep reflexes, a small and rapid pulse, dilated pupils, a rapid, superficial, or Cheyne-Stokes respiration, and various paralyses. Finally the power of deglutition fails, and respiration becomes stertorous, and the patient gradually sinks from exhaustion, particularly true in cases affecting the convexity, or expires in convulsions. Generally speaking, the symptoms of this stage

are of general cerebral disease, rather than of any localized lesion. We have just said that practically each stage lasts about a week, but cerebro-spinal cases sometimes die in two or three days, and are apt to run their whole course in a week. If they survive the first week, the case is apt to be one marked by remissions, and may last for weeks, or months, with variable symptoms, but conclusion in death is a vast probability. The possibility of remissions in the most desperate cases must always be kept in mind, but must not be made a basis for an optimistic prognosis. Meningitis in infants, while possibly fatal in a few days to a week (generally true of cases affecting the convexity), is most often a disease of many weeks, and may be months. Septic meningitis, and that secondary to infectious disease, is usually cerebro-spinal, and rapidly fatal, possibly in forty-eight hours. A meningitis following an abscess may produce death in a few days, whereas primary infection of the meninges, without the secondary process of abscess, may result in an illness which is fatal in a week or two, and in forms secondary to general disease it may run a course of several weeks. Tuberculous meningitis is peculiar in the long duration, and the insidious character of the invasion stage. This is followed by a stage of irritation lasting from three to ten days, and that of coma, which may last from a day or two to a week or two. Statistics show that one-fourth of the cases die in the first week, after the stage of irritation has set in (there may of course have been a long invasion stage), one-fourth die in the second week, one-fourth in the third week, and the other one-fourth during the next five weeks. Syphilitic meningitis furnishes the slowest cases, which may have an invasion period of six or eight weeks, and an irritative stage of irregular severity which may last months, or a year or two.

In general, one must expect a fatal issue, except in syphilitic cases, which are usually quickly relieved by anti-syphilitic treatment. There is almost unanimity in the opinion that tuberculous cases never recover, and that purulent forms are well-nigh as fatal. A small proportion of the Infantile, or Posterior Basilar cases recover, and a variable proportion up to 40% of the Cerebro-spinal type (variation is in the peculiarity and stage of an epidemic). Early cases in an epidemic are more apt to be fatal. The most quickly fatal are those purulent cases resulting from the rupture of a cerebral abscess. These are practically those which are spoken of in another place as instances of mixed infection. The Cerebro-spinal may be regarded as next in the rapidity of the process, while the Infantile are likely to be weeks, and perhaps months in duration, which period may be exceeded in some instances by Tubercular Meningitis. Recovery is never complete, and the patients remain defectives to a degree, all their lives. The most usual losses are in the functions presided over by the

cranial-nerves, and deafness, which tends to develop mutism, is the most common, unless strabismus surpasses it. On account of the cerebritis, which is to some extent a component in almost all cases, there is a diminution of motor or intellectual power in some or other part, and to a variable extent. The infantile case is almost certainly followed by some degree of internal hydrocephalus with its attendant damage to the functions, and future development of the brain.

DIAGNOSIS. The diagnosis in some cases is very easy, while in others it may be clouded by the intensity of the disease to which it is secondary, or the injury which has preceded it. In other instances the symptoms are fragmentary, as in the citation previously made, where cases of septic meningitis have been marked by delirium alone. If it is evident that the brain is organically affected, we are led to believe the condition to be Acute Leptomeningitis by the method of onset, and the manner in which the symptoms are combined, rather than by the presence of some striking phenomena. In the vast majority of cases, the onset is subacute, i.e., not a matter of hours, but of days, or a week or two in the slower forms of the disease. There is vomiting of a persistent and causeless type, a delirium co-existing with headache, and a headache which is not only severe initially, but persists, and is prominent, even when ordinary sensation has been abolished. This is an important point, since, as Jenner has pointed out in other diseases, however severe headache may have been as an initial symptom, it fades into the background as the delirium and general prostration become prominent. There is also an unusual relation between temperature and pulse, there are inequality of the pupils, strabismus and choked disc, nystagmus, weakness of the muscles of the face, retraction of the head; retention or incontinence are liable to be the conditions affecting the bladder and the rectum. Convulsions are apt to occur initially, or after the disease has become established. Ready blistering of the skin would be a confirmatory symptom, like the preceding.

Squires has found invariably present by the fourth or fifth day of the disease a rhythmical contraction and dilation of the pupil, when the head is placed between the knees of the examiner, face upward, with the body supported. Grasp the sides of the child's head with each hand, and produce gradual and forcible extension of the head on the spinal column. As the head is brought back in extension, the pupils will commence to dilate simultaneously with the commencement of the extension, and the degree will be measured by the degree of extension. Upon flexion the pupils contract, so that they are well closed by the time that the chin is forcibly brought to the manubrium.

BLOOD-CHANGES IN MENINGITIS IN CHILDREN. Leucocytosis means pus, and hence suppuration. In the estimation of the mor-

bid character of findings in children it should be remembered that while 5,000,000 red corpuscles in the male, and 4,500,000 in the female, with about 7,500 whites is normal in the adult in health, a different condition obtains in infancy. A child at birth shows about 5,749,500 reds and 15,000 to 20,000 white cells to the cubic centimeter.

At the age of one year we ought to find about 15,000, and at the age of seven we obtain the adult count of 7,500. The leucocytes are greatly increased after digestion, and at the outset are increased up to 30,000, and do not reach a normal until about five hours after the ingestion of food. If the food has consisted largely of fluids, or much fluid has been taken with it, there will be an apparent decrease of the red corpuscles. Diarrhoea, or other wasting diseases, will produce an increase in the count of both reds and whites. In tuberculous meningitis the reds show no change, or are slightly increased, and there is generally no leucocytosis. The eosinophiles are reduced, while the large lymphocytes, and transitional forms are increased. Normally, the eosinophiles are 1-2 to 4% of the total whites, and the large lymphocytes and transitional forms are from 4 to 8%.

DIFFERENTIAL DIAGNOSIS. Typhoid Fever. This, when affecting children, is much like in its onset, and the petechiae resemble those of the Cerebro-spinal form, but they are confined to the abdomen in typhoid, and this disease lacks the herpes labialis. The bowels are loose instead of constipated, swollen instead of retracted, and the initial headache ceases when delirium comes on, or if persistent, does not at any rate increase.

Febriculæ of children have headache and delirium, but they are initial symptoms only. The fever accounts for their presence, and the headache soon passes away, or fails to increase. Convulsions may occur in both these diseases, but they are initial, and are not presented in the same combination with other symptoms. Eye-symptoms especially are lacking, and, if they occur at all in febrile disease, are the results, and not incidents, of the acute stage.

Rheumatism. This may produce the rigidity of the neck, but the muscles are sorer, and while there may be considerable fever, the combined symptoms are lacking. This diagnostic error is, however, not a fanciful one, and the indications may be confusing at times. Urinary findings are helpful.

Muscular (nuchal) Rigidity from Other Causes. In other cases we may find this symptom, but it will be accompanied by swollen glands, and evidences of disturbance of abdominal or other organs, or an angina.

Intra-cranial Tumor. Onset very slow. Chronic. Limbs slowly grow weak, optic neuritis is severe, temperature and pulse are undisturbed, or only intermittently so.

Hydrocephaloid of Marshall Hall. This has the symptoms of acute hydrocephalus from a congestive meningitis, but there is the history of, or existing, exhausting disease, the fontanelles are depressed, and there is no nuchal rigidity.

Hysteria. This may simulate, but organic signs are always wanting.

Meningeal Hemorrhage. This is very rare, but at times closely resembles it, since both diseases are subacute in onset, both have conspicuous delirium, and both follow an injury. Fever, however, is lacking and after similar invasion symptoms, the actual invasion is quite likely to be apoplectic.

TREATMENT. In all cases the patients should be placed in a cool room, darkened and removed from all disturbing noise. They should be guarded from all unnecessary visitors. In the early stages they are benefitted by ice-bags to the head and neck, and heat to the feet. Mustard to the feet may give comfort, and also may be of real benefit. Warm baths are useful, and Aufract advises a bath of ten minutes duration at a temperature of 100°, and as many as eight have been given in one day with benefit. In all cases where meningitis is secondary to some focus of infection all avenues ought to be kept in a sterile condition. The sterilization of the intestinal tract does not seem to be a possibility. Counter-irritation along the spine is not in favor, on account of the liability of sloughing of the skin in this disease. If vomiting is a feature, feeding should be rectal, and in any case, and by any avenue, must be frequent, and in small quantities. Stimulants are not well borne in the stage of excitement. Efforts to relieve the vomiting should be such as will not be troublesome to the patient, since they are usually ineffectual. In the stage of stupor the head should be kept high, and flexion of the neck should be avoided, since it interferes with the easy return of the blood from the head. In this stage full feeding has a positive therapeutic effect, and should be carried out by the rectal method, or the stomach or nasal-tube. It has been insisted upon by some authors that a broad fly-blister on the nape of the neck, reaching from one mastoid process to the other, would arouse the patient from the stuporous condition, and act with curative effect upon the causative condition, but this position has been specifically denied by men of equal experience. Whiskey, or more specific stimulants, are advised in this stage. Applications of Iodoform ointment (see treatment of tuberculous tumor) to the shaven scalp (which shaving, by the way, should always be the rule). Crede's Colloidal Silver ointment, Tartrate of Antimony ointment, Mercurial ointment, all seem to have done good in occasional cases. Trephining and ventricular puncture have had advocates, but no general endorsement. Lumbar puncture was introduced by Quinke, and has been used with increasing frequency as time

has developed its value. The theory is, that by the process we are put in possession of a knowledge of the constitution of the cerebro-spinal fluid, and will be able to diagnose the variety of meningitis with which we are contending, and also have in our hands a means of diminishing the evil effects of the compression of the brain. This latter was the first effect that was sought. To these two were later added the hope that following the removal of the excess of the fluid, a bactericidal agent could be introduced which would tincture the whole of the cerebro-spinal fluid, and thus specifically attack the disease at its source. The agent most vaunted was 2% lysol, and later, in the epidemic cerebro-spinal cases, diphtheritic antitoxin. Neither of these has proved to have any curative influence upon any form of meningitis. The reduction of pressure is certainly demonstrated, and that it does relieve the stupor and delirium. The pressure returns, but as the operation can be many times repeated without injury, it is a valuable method for the relief of this condition. Diagnostically it is extremely valuable, since it shows by the abnormal force of the flow the amount of intracranial pressure, for in health the flow is drop by drop at intervals of one to several seconds. The normal fluid is clear as water, and contains the merest trace of albumin, if any. In meningitis it is cloudy, flocculent, and albumin is increased to the extent of 2%, or more. By the microscope we can detect the character of the micro-organisms producing the condition. The technique is as follows: place the patient erect in a chair (if a child, double up in bed while lying on the side), and have him bend as sharply forward as possible, so as to produce the greatest possible curvature of the spine in the lumbo-dorsal region. Make the skin sterile, and then gradually insert one of Quincke's needles, or a very fine trochar, in the center line between the spines of the vertebrae, in a child, and direct it upward. In an adult 1-2 to 1 inch to one side of the center-line, and direct the needle upward and inward. This, in both instances, should be inserted on the level of a line drawn between the iliac crests, which makes it between the two upper lumbar vertebrae (some say it is better between the 3d and 4th lumbar). No anesthetic is needed, although ethyl chloride may be used. Incising the skin previously with a bistoury takes away much pain. Push the needle in slowly, until resistance suddenly ceases; this will occur at a depth of 2 c.m. in a child, 6 c.m. in an adult. Never use suction. From a drachm to two ounces may safely be withdrawn, depending upon pressure. Vomiting and collapse have occurred, but are rare or trivial, unless an uncalled-for amount has been withdrawn. Possible bad results are cardio-respiratory disturbances, asphyxia, panting, nausea, vomiting, sweating, cyanosis, collapse, rigors, cramps, transient paraplegia, sensations of heat, or of general depression, elevated

or subnormal temperature, fibrillary muscular contraction, relaxation of the sphincters, retention of urine. The needle should be 3 inches in length.

THERAPEUTICS. The treatment must be largely symptomatic, in the sense that no specifics are known, nor drugs approaching such a degree of potency.

Stage of Invasion. Begin with Aconite in a sthenic case, and persist in its use up to the period when signs of compression begin to show themselves, or paralysis becomes evident. All strengths of the remedy have been advised, from doses of 1-10th of a drop of the tincture for an adult, and 1-100th for an infant, all the way up to the highest potencies. There is no darkness of the skin, but the face is red and flushed, pulse quick and strong.

Belladonna is preferable when the excitement is still higher in grade, and symptoms point specifically to an invasion of the cortex. The patient is especially free from depression, there is no darkness of the skin, but the face is red and flushed, and the pulse is quick and strong. Acuteness of onset is diagnostic, and while there is wild cerebral irritation, there is none of the fidgetiness of Apis. Typically, a child may have gone to sleep well, awakens later with hot head, cold body and extremities, cries out and has spasmodic jerking of the limbs.

Gelsemium has been given the precedence, by some, over the two preceding remedies, especially in the place of Aconite, in those cases which have begun with rapid talking, and a quick nervous manner. Especially in the cerebro-spinal form, but its value here is only for a very short space of time, as the next stage comes on very rapidly.

Veratrum vir., in tangible doses, was strongly advocated by Hale (drop 1-100th to 1-10th), and alternation with Hyoscyamus and Belladonna is suggested, if the pulse and temperature remain high. Indications are great irregularity of superficial temperature, vomiting, great aggravation of head from raising it, proportionate ease from lying, the least motion causes increase in nausea and vomiting, considerable unsteadiness in the extremities, rigidity of the neck, scowling and pallor, and flushing of the face upon one cheek only.

Bryonia is preferred in the Tuberculous form, but it is not here a remedy for the initial stage, yet this is usually so ill-defined that the case is not often truly appreciated until the second stage has been reached, and a proper interpretation of the symptoms points out that effusion has already occurred.

Stage of Irritation. Belladonna is advised by some in this stage also. Agaricus is advised in the 3x dilution for delirious cases. Hyoscyamus is very useful also in mental exaltation, and the delirium is active. In the midst of a perfect stupor the patient reacts to the slightest stimulus. There is mania, which is relieved

by violently shaking the head. Stramonium in wilder cases in the same dilution.

Bryonia when the temperature falls, and signs of effusion come on with a falling pulse. It is held by some to be the best remedy. Jahr advises it when the meninges, rather than the cortex, are the seat of attack (the opposite of Belladonna). Excessive pain and sensitiveness of the cranium are indications for its use. The pains are sharp and sticking, and there is a constant chewing motion. Pains are aggravated by motion. The face is dark and congested, the lips are parched, drink is taken hurriedly and impatiently, and the child shrieks with pain. The type is a rather asthenic one, and covers all the symptoms clear up to the developement of the petechiae. The delirium is rather mild, but the pain is excessive. It is especially well indicated after Aconite, and, like it, is of no value after the paralyses have become evident. There must be a livid and flushed face, high temperature, an easy sweat with nausea, desire to vomit, and constipation with *distended* abdomen, and scanty and painful secretion of urine. It is advised in the 2x.

Apis after secondary infection from the exanthemata with immediate signs of effusion, and a markedly scanty urine. The case begins with sharp cries and fidgetiness, and there is a perversion of the senses. In cases where there is a restlessness of one half of the body, and a hemiplegia later. It follows Bryonia peculiarly well, and is for those cases with evident effusion where Bryonia has failed to relieve. Rhus has similar symptoms, but there is great bodily restlessness, instead of the mental which is found in Arsenic. Strongly recommended.

Zinc may be of use in this stage where symptoms of irritation exceed those of compression. The child awakens in fear, rolls its head and cries out, complains of a sharp, sticking pain at the root of the nose, and in all the branches of the fifth nerve, and is worse from stimulation. Some authors feel that the potency of this remedy is much over-rated.

Cuprum, either metallicum, or arsenicosum, is extremely beneficial in the irritant stage. The child screams loudly, its fists are clenched, convulsions are violent, the face is pale, the lips are blue, and the eyes are oscillating rapidly. One of the best remedies.

Asthenic forms, where there is little reaction, cause us to think of Arsenic, Rhus, Hellebore, and the petechiae are also indications.

Iodine is clinically well supported, but the symptomatological indications are meagre. Rhus may be used in frequent doses of the lower dilutions.

Stage of Compression. Opium when the patient is comatose, or in stupor with convulsions, with incontinence of urine and faeces, and in the last stage with contractions of muscles. Hale advises the use of this drug in the 2x dose, a grain every two hours. The

author has seen good effects in well-marked conditions from the 200th. It is advised for a coma which does not yield to *Belladonna* or *Hyoseyamus*.

Hellebore is advised in the last stages with falling temperature. The forehead is wrinkled, the lower jaw drops, pupils are dilated, and do not react to light, there is automatic action of one side, there are complete mental apathy, with no response to touch or pressure, dark sooty nostrils, and a slow pulse with cold sweat. The value of this remedy is very much in doubt.

Actea racemosa when convulsions have persisted after the active symptoms have subsided.

Iodoform, in the tuberculous form, a tablet of the 2x every two hours, is as good as the use of the ointment.

Kali iod. In tangible doses in the chronic forms. *Sulphur* is good as in other diseases as an intercurrent.

Mercurius is to be given in the early stages where we would think of *Belladonna*, but the throat is sore, and the glands are swollen.

Belladonna. In its place *Atropia 3d* may be given in solution every one or two hours.

Strychnia. Dr. J. T. Simonson of New York reports the successful treatment of several cases of the cerebro-spinal type by the use of this remedy in the sixth potency. It was given in cases of great severity which displayed the following symptoms. Very severe occipital pain, extending into the cervical region and often down the spine, with marked tenderness to touch. *Opisthotonos* marked. Extreme hyperesthesia of the special senses and along the distribution of the spinal nerves. Respiration was irregular in force and rhythm. Tonic muscular contractions marked, especially in the back. Drowsiness, constipation, nystagmus, spasm of pharyngeal muscles on attempts to swallow, choking sensations.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

DEFINITION. A form of inflammation of the meninges of the brain, and the spinal cord, which usually occurs in epidemics, which at a given time extend over a large portion of the country, but in a very irregular way. It does not attack a large number of people in any locality, but in small scattered groups. Epidemics vary very widely, both in virulence, and in universality. It does not seem to follow lines of travel. It acts as if there were a material source of infection distributed through the air during the initial stages of the disease.

DIFFERENTIAL DIAGNOSIS. It is to be differentiated from Tuberculous Meningitis, Typhoid Fever, Uremic Coma, Rheumatism, Hysteria.

SEX. This is not a factor.

AGE. Children, from one to ten years of age, furnish the greatest proportion of the cases, and it formerly was considered that persons over thirty were almost immune, but this has not been true of recent epidemics. In the epidemic of 1905, many persons in late adult life, even to the age of sixty, were victims of the disease.

CLIMATE. It is a disease of temperate and subtropic zones, without narrower limitation; and while poverty, privation, and unsanitary surroundings furnish, as in every other epidemic condition, the greater proportion of patients, the opposite of this does not confer immunity. It seems to be most common in the spring months of a cold and wet year.

ETIOLOGY. It is a disease arising from a definite infection, but not from any single micro-organism. The efficient agents appear to have been the diplococcus meningitidis intracellularis of Weichselbaum, and the pneumococcus of Friedlander, although many other micro-organisms have been found in combination with them in the cerebro-spinal fluid. Osler states that the diplococcus of Weichselbaum is the cause of the disease, when it is confined to the meninges, while the pneumococcus is the agent when meningitis is merely one element in the general condition. In a large proportion of the cases, it is probable that the infection gains entrance to the body through the nasal tract, although some feel that invasion may be by the gastro-intestinal route. The former belief is founded upon the fact that there is usually an antecedent influenza of a purulent type, and the diplococcus of Weichselbaum has been demonstrated in the secretion.

COMMUNICABILITY. The investigations of the New York Board of Health have proved that the meningococcus can be found in the nasal secretions in the majority of cases in the first week of the disease, but not frequently after that time. It seems to be communicable to those in direct contact, through this agency. The micro-organisms have a very feeble power of resistance, and can be almost instantly destroyed by the action of thirty per cent. alcohol.

PATHOLOGY. The essential process in this disease is an inflammation of the Pia-arachnoid of both the brain and the cord, and the result is an infiltration of this membrane, with a rich cellular migration from the vessels, resulting in an effusion over the whole surface. This fluid is turbid with cells, and swarming with bacteria, and contains an excess of fibrin. The effusion compresses the brain and the cord, and the fibrinous increase tends to bind the membranes together, and compress the emergent nerves, both cranial and spinal. It often invades the ventricles, and by occluding the foramina, and inflaming the ependyma and choroid plexus, sets up a condition of hydrocephalus. As in other forms of men-

ingitis, the change in the structure of the Pia interferes with the whole circulation of the brain, and, in this case, of the cord also, and results in all stages of inflammation and softenings, of greater or less extent, so that here we are dealing with the results of an inflammation of the meninges, accompanied by more or less cerebritis, and myelitis, and, from its origin, the latter will be of the marginal variety, similar to that arising from spinal syphilis.

SYMPTOMATOLOGY. The clinical picture is much like that of the forms of meningitis just described. They show large variations in distribution and severity of the symptoms, and this form is no exception to that rule, since different epidemics possess a widely variable degree of virulence, and in any epidemic the later cases are invariably less severe. With all the variations, however, cases reasonably conform to the following description:

The onset is marked by a severe headache with vomiting and vertigo; the patient is sensitive to any light or noise; he is tormented by frequent chills; and, especially in children, there is great liability to convulsions. The patient then becomes restless, but, in spite of all, with a clear mind. Initially, or within 48 hours, the neck becomes stiff to some degree. Temperature and pulse begin to rise, but neither one shows any diagnostic features, except that with the usual temperature of 102° at this period, the pulse is rather low in frequency.

The spleen is enlarged, but not always so as to be discoverable by palpation. As the disease develops, the headache still remains severe, or increases; the mind now has become clouded by delirium, and the nuchal rigidity has become more pronounced, and the spine, the muscles of the trunk, and extremities show the same condition. Finally, the head becomes bent back until the occiput lies between the shoulders; there is arching of the spine, so that the body rests solely upon the head and buttocks (*Opisthotonos*), and the muscles of the abdomen are so rigid that the abdomen is flattened, or even concave (*Boat-shaped abdomen*).

Hyperesthesia of the surface is so great, that touching the skin, or pressing upon a muscle will induce such a degree of pain that the patient will scowl, or cry out, even in his dull mental conditions. There is, from the beginning, a drawing pain in the muscles of the extremities. The initial constipation, and retention of urine are liable to be replaced by incontinence in the later stages, as in other forms of meningitis. The urine shows albumin, and sometimes casts. The face becomes pale and pinched, the pupils are widely dilated, and the ophthalmoscope will usually reveal a beginning optic atrophy. This status is gained usually by the second or third day, and by this time the eruption, which is not invariable, but a common feature, has become evident. *Herpes labialis* is one of the symptoms which might be called diagnostic. It comes in crops, lasting only a few days. In addition, there are

also petechiae, diffused over the entire body, but more deep and purple in some localities. Urticaria and purpura may also be found. This cutaneous irritability is, with the added element of vasomotor paralysis, the cause of the "Tache Cerebrale" of Trousseau, which was formerly considered diagnostic. This variety of trophic disturbance may be intense enough to cause bedsores. The cranial nerves are affected in almost every case, and ocular motor palsies common, producing strabismus, and facial palsies are fully as frequent. The membranes and structures of the eye are apt to be the seat of all grades of inflammation, even up to a destructive grade. Chemosis is almost universal. The delirium by this time has become very severe, and is worse at night, and varies in intensity with the degree of fever present. The mind is so clouded that cases which recover have little recollection of their sufferings. General, and partial convulsions are liable to occur at any stage, and in the fully developed cases there may be monoplegias, and even hemiplegias.

At times the joints will be the seat of an exudation which contains the specific micro-organism, and may be purulent in character. This may be not a complication, but a sequel of the disease.

In place of the cranial nerve palsies in the region of the bulb, we find root and girdle pains in the distribution of the spinal nerve. The knee-jerks are abolished, the bladder is paralyzed, and the muscles of the neck will be found to be powerless if the case proceeds to recovery. While this description will fairly portray the symptoms of an ordinary case, we may find cases which prove fatal in from a few hours to a day or two, whereas others will show a protracted form, so intermittent that it appears almost malarial in its causation, and the patient may die from exhaustion, bedsores, etc. Then there are abortive forms, which start with great severity, but never develop into the typical disease. There are epidemics, where the superficial symptoms are entirely unlike the classical ones which have been mentioned. Dr. Samuel Gordon, in an epidemic in Dublin in 1866-67, noted that the patients had a coldness, and blueness of the skin like cholera; bruises and ecchymoses like scurvy or typhus fever; hemorrhagic eruptions over the whole body, chiefly on the lower limbs, which were dark brown, purple, or black in color, in large or small spots, which were flat or elevated, and they might become gangrenous; hemorrhages from the mucous surfaces, nose, mouth, stomach, bowels, and kidneys.

PROGNOSIS. Fatal cases will show a coma, which does not remit, or which becomes deeper; there is progressive emaciation, the sphincters are paralyzed; there is profuse perspiration, and meteorism. The temperature may go as high at 108°, or fall to subnormal, and the pulse is small and frequent, and intermittent.

In such cases death will occur at the end of the second, or beginning of the third week.

There is another class of cases, which is not fulminant, nor so severe as to seem hopeless, but improvement is only remission, and, after repeated improvements and relapses, the patient finally dies of exhaustion. There is no disease where certainty of prognosis is more difficult, so far as mere preservation of life is concerned. The most intense cases will sometimes end in recovery even after the appearance of symptoms which would be considered unequivocally fatal; and on the other hand, convalescence must be very complete before we can feel certain that it is not merely a very prolonged remission. The favorable cases show it from the first by the slightness of the symptoms. There is either no coma, or it is quite incomplete, and the temperature falls, or becomes intermittent, and by the tenth day the restlessness decreases, and the appetite returns. Such a case, however, may have a very protracted convalescence. On account of the contraction of the exudate, which has been poured out over the different parts of the brain, the bulb, and the spinal cord, the occlusion, partial or complete, of the foramina of the ventricles, we should expect some grade of hydrocephalus, some degree of change in the cortex of the brain, and a detrimental infiltration about the cranial and spinal nerves. From all these causes, the patient's recovery is usually very incomplete, and some symptoms may remain for a very long time, or become permanent. There is a lifelong tendency to headache, and the disposition is often changed, and in many there is a considerable mental reduction. Tinnitus aurium, and strabismus are almost universal; less common is blindness from optic neuritis, or optic atrophy, and from changes in the globe of the eye, but rarely from degeneration of the visual centers. The prognosis is bad for recovery. Deafness is very frequent in the first two weeks of the disease, less common in a later stage, but some proportion of the cases will be permanently deaf, which in its turn tends to produce deaf-mutism. It may become a permanent deficiency, even after abortive types of the disease. If it persists after three months, it is usually permanent. Another residual symptom, which is especially marked in children, is such an amount of ataxia, that the patient sways in walking, and from which it may take years to recover. From the spinal invasion we may get paraplegia, or paralyzes of the spinal type, long lasting, or even permanent. The tendency of the hydrocephalus is to permanently compress the brain, and produce secondary atrophy. The symptoms indicating this condition during convalescence are headache, vomiting, attacks of coma, and recurring convulsions. Recovery from this condition is doubtful. In convalescence, the patients are very prone to joint disease, pneumonia, and phthisis, and pyæmias. The prognosis will be more doubtful,

if concurrently with the meningitis there is a croupous pneumonia, scarlatina, pericarditis, etc. In general the disease is rapidly fatal, with death in from five to eight days, and more in the period from the second to the fourth. The mortality is 40 to 50 per cent., being as low as 20 per cent. in some epidemics, and as high as 80 in others.

DIAGNOSIS. The essential symptoms are a meningitis of an epidemic character, with conjoined cerebral and spinal symptoms, marked by early opisthotonos, and cutaneous eruptions, especially herpes labialis.

DIFFERENTIAL DIAGNOSIS. Tuberculous Disease. This lacks the herpes labialis, and, in the cerebro-spinal fluid of the epidemic form, we do not find the tuberculous bacillus. We also have diarrhea in place of the constipation which is usual, or the dysentery which may occur.

Typhoid Fever. This may resemble it very closely in symptoms, and both are marked by enlargement of the spleen, but lumbar puncture will differentiate it. Lacking this, the typhoid temperature is step-like in its increase, the stools are loose, meteorism is almost always present, and it is early in appearance. The abdomen is bloated, rather than retracted. Muscular rigidity is evident in the neck in both diseases, but in typhoid fever it does not extend to the trunk and legs. Tetanus and muscular rigor may be present, but the associated symptoms are lacking.

Uremic Coma may display convulsions, there may be vomiting, and nuchal rigidity, but there is no fever, nor herpes, nor hyperesthesia. We cannot base a diagnosis upon an examination of the urine, since both conditions may be marked by the presence of albumin and casts, and this point would only be elucidated by a knowledge of the previous history.

Rheumatism. This would show a swelling of the joints as an initial symptom, there might be rigidity of the neck from resisting attempts at movement which might cause pain in the muscle, and the same might be true of the back, but there is no similarity in the associated symptoms.

Hysteria. During an epidemic of Cerebro-spinal Meningitis, hysterical persons often show a rigidity of the neck, but associated symptoms will show that it is a disease of the mind, and not of the spinal meninges, and the same is true of opisthotonos, occurring under the same conditions. On examination we should find, also, that there is no fever, nor paralysis of the cranial nerves.

TREATMENT. For general therapeutics see the preceding article. See Strychnia particularly. The Flexner serum seems to be the most successful means of combatting this disease. It is injected into the spinal canal, following lumbar puncture. The amount administered should be about 45 c.c., following on the abstraction of an equal, or greater amount of cerebro-spinal fluid. It should

be instilled very slowly, in order not to raise intra-cerebral pressure. It may be administered daily, so long as demanded by the disease, and the initial injection may be given before the diagnosis by cultures can be made, inasmuch as its uncalled-for administration is harmless.

CEREBRAL APOPLEXY FROM HEMORRHAGE.

DEFINITION. It is that combination of symptoms which results from the rupture of an artery in the Cerebral Meninges, or in the substance of the brain, or brain-stem. It results in a motor or sensory palsy (or combination of the two), of greater or less extent, and secondary motor changes resulting from subsequent degenerations; mental changes probably.

THE SYMPTOMS OF A TYPICAL CASE. There is sudden unconsciousness lasting for two or three days, paralysis of the whole body, and a suspension of all the functions, except those of the heart and lungs. The pulse is strong, full, and slow, and the respiration is stertorous. In two or three weeks the general paralysis has become a hemiplegia, and the patient later regains a moderate degree of health with weakness and contracture of the paralyzed side. He dies after two or three repetitions of the attack.

DIFFERENTIAL DIAGNOSIS from Syncope, Toxemias, Poisons, Alcoholic Intoxications, Uremic Coma, Meningeal Hemorrhage, Epilepsy, idiopathic and focal, Hysteria, General Paresis, Diabetes, Acute Softening from Thrombosis and Embolism, Fracture of the Skull.

AGE. While it is possible at any time of life, it is rare before the fortieth year, and is most frequent in old age.

SEX. Males more often than females.

ETIOLOGY. This varies somewhat according to the site. Meningeal hemorrhages causing apoplexy are the result of injuries to the cranium, and in such a case demand no antecedent disease of the arteries to make them possible. They may also result from anything which gives rise to an increase in the intra-cranial pressure, as in the well-known instances where it has occurred from the straining at stool. In such case some antecedent condition is demanded which shall have weakened the coats of the arteries. This may be due to some pre-existing disease, or have been the result of Senile Degeneration. More rarely Hemorrhage occurs from a laceration of the veins or sinuses. This is due to violence. In the cerebral type it may be due directly to the diseased condition of an artery, or to a strain upon a weakened artery. That it is only a terminal phenomenon of a general and long-standing condition is proven by the fact that autopsy almost

universally shows that in such cases the cerebral arteries are studded with numerous, and widely disseminated dilatations, any one of which was the site of a potential rupture. The predisposing causes are old age, intoxications (lead for example), infections, gout, nephritis, and also heredity.

PATHOLOGY. In 60% of the cases it is due to the rupture of the lenticulo-striate, and lenticulo-optic arteries, and most often those upon the left side of the body. This localization comes from the fact that they are largely terminal arteries, branches of the middle cerebral, which is the direct extension of the internal carotid, and are peculiarly vulnerable upon the left side, for the reason that while the right common carotid is given off from the right subclavian at right angles, thus checking the hydrostatic pressure of the blood-column, the left rises in a direct line from the left innominate, which is equivalent to an origin directly from the arch of the aorta. The full impact of the contraction of the ventricles is thus felt more strongly than elsewhere in the lenticulo-striate arteries. Leaving out of account the meningeal attacks, the localization in order of frequency is capsulo-ganglionic, cortical, ventricular, subcortical, centrum semi-ovale, cerebellar, pontile, oblongatal, crural.

At the site of all these accidents we find some variety of chronic endarteritis. By this is produced a patch of atheroma, which later undergoes fatty degeneration, which breaks down from the completeness of the process, or from temporary overstrain. This does not affect the small arteries of the brain. In senile conditions the arterial walls may become thinned out without evident disease or pathological change. After the rupture there is a difference in the results from the variation in site. If it is cortical the extravasated blood spreads out in a thin layer, and may be multiple. It may not be absorbed or organized for weeks, while if subcortical it tears the soft brain tissue, or it may rupture into the ventricles, where it may consolidate into a perfect cast of the cavity, or following the iter gain entrance into the soft tissues of the fourth ventricle. As a general rule it tears out a space of about two inches in diameter in the soft tissues of the brain. The clot may be as large as one's fist, or as small as a hazelnut. All about this area the brain tissues are for some little time in a state of compression. Very soon this surrounding space becomes edematous, and is the site of numerous capillary hemorrhages, on account of the changes of blood pressure, and local inability to resist it. All these changes combine to produce the initial state of unconsciousness, shock, and universal paralysis which mark the early days of an apoplectic attack. The shock is produced by the direct pressure of the clot (Niemyer); only in part says Gowers, who attributes the almost universal period of unconsciousness to the resulting interference with the higher functions of the brain.

However, it actually produces the effect. Tests have shown that a pressure of 130 mm. on the surface of the brain of a dog will render the animal unconscious, while one-tenth of that pressure will produce the same effect if exerted suddenly. Slow exudations are compensated for by a relative decrease in the cerebrospinal fluid. The clot begins to lose its bloody color in from two to five weeks, but remains hard for about four weeks. In all cases by the fifth week it is yellow, as a result of the activity of the leucocytes. These cells, enclosing blood-corpuscles become evident about the third day. From the fifth to the twelfth week it is a yellowish mass, and then becomes ocherish in color, from the presence of free pigment. While after a small hemorrhage a cyst-wall may appear in a month, and contraction may begin a little later, it more often takes from three to six months. This cyst-wall is formed from glia and connective tissue, which begins to contract possibly in a month and a half, but often a year elapses before contraction, so that a spot is not actually quiescent until a year, or a year and a half to two years from the date of the apoplexy. This cyst may undergo fatty degeneration, and replacement by fibrous tissue, or it may remain a cyst, filled with straw-colored fluid. Obliteration is rare after hemorrhages of much size. This area of changing tissue is essentially a foreign body, and, by its pressure, may more or less completely inhibit the function of the surrounding portions of the brain. The fibres descending from injured motor cells begin to degenerate at the 10th to 14th day, and in old cases the degeneration has been found to extend down the cord. As in all other lesions of nervous tissue, the replacement is by structures incapable of nervous function.

SYMPTOMATOLOGY. Premonitory symptoms are rare, not being like thrombosis, where we may see for a period of hours or days some slight dizziness, fullness in the head, bad dreams at night, headache with epistaxis, disturbance of heart's action, and a little numbness of the hand or foot upon one side. Occasionally we do see definite symptoms and they then arise from an exaggerated degree of arterio-sclerosis, and differ from those just given in the fact that they are of more gradual onset, while those of thrombosis are present for a much more limited period. The only source of error would arise in cases like that quoted by Hardy, where there was a headache for ten days, and then a hemiplegia. The autopsy showed that there had been a hemorrhage in two stages, both of them occurring without loss of consciousness. The rule is, however, that before the attack the patient is often remembered to have said that he was feeling unusually well, and the rule of the attack is pre-eminently loss of consciousness, followed by hemiplegia. Pain in the muscles of a whole side have preceded an apoplexy for from twenty-four to forty-eight hours. In a typical

case, which is generally the fact, and typifies the popular conception of the word "stroke," the victim drops in unconsciousness, with unequally dilated pupils, which do not respond to light. The deep reflexes are exaggerated for a few moments, and then obliterated, together with the superficial ones. The heart is tumultuous for a few moments, but after a little time the pulse is tense, full, and slow. The temperature is normal, or subnormal. Urination and defecation may occur, and swallowing is impossible. Isolated cases may occur, where the patient runs a few steps in an aimless manner, grasping the head, and cries out as if conscious of an injury to the brain, but this is more common in embolism. At times a patient may grow confused, complain of palpitation, numbness in a part, loss of sight and hearing, become thick in his speech, have relaxation of the sphincters, and then develop complete coma. A convulsion may be the initial symptom, but some, or all of these symptoms will be present in every case. Breathing is stertorous and slow, and will become Cheyne-Stokes in rhythm, if a fatal issue is impending. If this persists for more than three days, mucus will collect in the lungs and trachea, and death will follow from the results of this; i.e., Inspiration Pneumonia. The face is congested and bloated. From the first there is a disposition to fix the eyes and the head (conjoined deviation has a common center in the cortex) toward the lesion, and away from the side which has suffered the paralysis, but the contrary is the fact if the lesion is in the pons. In analyzing this symptom we must be careful to distinguish it from the spasmodic twitching which may be present, which is an irritative symptom. This conjoined deviation is a temporary symptom, passing away as consciousness returns, and is due to the unopposed action of the unparalyzed muscles. For a day or two the urine is increased, and for the first twelve to twenty-four hours contains albumin or sugar. According to the severity and extent of the hemorrhage, this group of symptoms will endure from an hour or two to three or four days. During this period it is often difficult to determine which side of the brain is the seat of the hemorrhage, since at this time the whole body is the seat of a general flaccid paralysis, but, by the exercise of care, it may be discovered that one side is more limp than the other, and that if that member be lifted from the bed it will fall more heavily than the other. Where diagnosis of the side paralyzed is difficult, examination of the abdominal and cremasteric reflexes will disclose their absence on the side paralyzed. Another test is by placing the limbs in an uncomfortable position. After a time they will be adjusted more comfortably upon the well side, but will lie unmoved on the paralyzed one. After this initial period of shock, the coma will lessen, and the well side will begin to show evidences of power. Deep coma is replaced after some hours by the lighter grade of unconsciousness which we term

somnolence, and in this state the corneal, and other reflexes become evident again, the patient can swallow, and execute some movements of a slight nature on the well side. If consciousness does not return in two or three days, the shock to the brain has been so great that the nutrition is sure to suffer, and bedsores will occur. The pulse and temperature will now begin to rise. The initial fall of temperature was from a little below normal down to 97, or 96 even, and the subsequent rise is apt to begin in from three to twenty-four hours. It soon attains a height of 103 to 104 degrees; if it does not stop at this point, but goes on toward, or up to 107 or 108 it becomes a sign of a complicating pneumonia, and a continuance of the subnormal state points just as certainly to a fatal issue. The skin now becomes moist, and headache begins to be evident; there is some delirium. There are all grades of this condition, but by as much as the lesion is removed from the cortex, by just so much is the disturbance of consciousness lessened, and the resemblance to a spinal lesion increased. Vomiting may be a symptom, and is more apt to occur as the lesion approaches the cerebellum, or bulb, or invades the ventricles. The paralysis will vary in extent and severity according to the location and amount of the causative hemorrhage, but it is always a hemiplegia of the face, arm, and leg type, and even when the whole body is paralyzed it will be by a double cerebral lesion or ventricular hemorrhage, in either case resulting in a double instead of a single hemiplegia. Speech is notably affected if the paralysis is of the right side, since it will arise from a lesion of the left hemisphere, which is the side upon which speech is developed, unless the patient is left-handed. It is either a dysarthria from paralysis of the lips, tongue and cheeks, or of the finer muscles of articulation, or it is an aphasia from destruction of specific centers, or of the association tracts. Specific localizations for varieties of aphasia have recently been questioned. Hemorrhage in the central ganglia of the left side, or in the internal capsule of the left side, will cause it, and the more extensive it is, and the nearer the approach to speech centers, by so much will the aphasia be marked and permanent. Exceptionally a paralysis may always remain flaccid, but usually a rigidity ensues sooner or later. Rigidity is of two types: viz., early and late. Early rigidity may come on temporarily in the reaction stage, and is a sign of irritation from secondary inflammation, rather than of destruction of the motor cells. Swallowing is impossible in the stage of coma.

When the reaction which has just been described is fully accomplished, symptoms of a more permanent character begin to be evident. The condition is now a hemiplegia of some definiteness. On examining the face one will notice that the expression is changed. The lines are smoothed out, and the eye is more open

on the paralyzed side, the corner of the mouth droops and the saliva is not swallowed, but dribbles from this side of the mouth. Raise a member on this side of the body and it falls limp, showing the absence of power in the muscles, but they may still be the seat of irritation displayed by twitching of the limb. The superficial reflexes are still absent, but by the second day the deep reflexes will show signs of the exaggeration which will be present permanently, or at least for a long time. It is possible that there will be a "crossed knee-jerk," i.e., on tapping the patella of the paralyzed side the other leg will be jerked forward. Ankle-clonus becomes evident, and after the first few hours there is Babinski's reflex, i.e., on stroking the sole of the foot there is extension of the great toe, and less often of all of them. This will be permanent. On account of the fact that the members upon one side of the body are represented to some degree on both sides of the brain there will be a weakness of the well side of the body, and the reflexes will be to some extent exaggerated. The paralyzed side will at first show diminution in the appreciation of touch and the other sensations up to a complete anesthesia, but, unless the lesion is in the posterior third of the internal capsule (which is the sensory division), it will pass away as consciousness returns. Hemianopsia (blindness of right or left half of both eyes) is at first more commonly present than is supposed, but it passes off when consciousness returns, unless the visual path is actually involved in the lesion. When hemorrhage involves the path of the fibres of any special function or sense, such as hearing, taste, or smell, we shall find a like loss of that sense. If a lesion of the motor area is incomplete, and therefore irritative, we shall find tremor of a rhythmical or intention type (like that of multiple sclerosis), ataxia, athetosis, or choreiform movements. In such cases the lesion is certainly in the upper motor neuron, and probably in the basal ganglia, especially often in the optic thalamus, or is such that it impinges upon the motor fibres in the internal capsule.

After a few weeks the paralyzed limbs begin to be capable of some useful amount of motion, more on the flexor than on the extensor side, and the legs gain more rapidly than the arms on account of their more complete double representation in the brain, and in all grades of recovery the legs retain this preponderance. The leg can generally be drawn up by the second or third week, and in cases of a slight degree of severity, the patient can walk in from a month to a year. The face also gains more than the arm for the same reason, that it also has a double representation in the brain. It may regain its power of expression in two months. At the outset the entire half of the face may be paralyzed, but, except in the rarest cases, the lower half of the face only is involved after the first day or two. The mouth will be wrinkled and the eye closed, as well as the

upon the other, or with only a slight degree of incapacity, but while attempts at voluntary motion increase the deformity, it will react normally to the play of the emotions. The only exception to this rule is when there is a definite lesion of the emotional motor centers (the basal ganglia, particularly the optic thalamus). In all these instances, however, the normal condition of the forehead and eyelids proves that it is a central loss, and that it is not due to a lesion of the 7th nerve, or of its nucleus, in which case the upper face is as immobile as the lower part. The arm will therefore, for the reasons just enumerated, show the slowest and least perfect recovery, and the finer motions of the hands last of all, if they are ever restored. Motion of the eyes is not affected, after the initial conjugate deviation previously referred to, unless the lesion involves the crus or corpora quadrigemina. Lesions in the pons will cause a defect in articulation like a motor aphasia. Secondary degeneration of the motor fibres begins in from ten to fourteen days after the apoplexy, and in one to four months we find what is termed late rigidity. This late rigidity sets up changes in the muscles which produce contractures, which are more evident in the arm than in the leg or face. Contractures in the leg are in extension; contrary to the rule in spinal lesions; flexion being found only in patients who are bedridden. In the upper arm there is adduction, while the lower arm is flexed with the hand pronated, and slightly flexed. The proximal phalanges are somewhat flexed, while the middle and distal ones are strongly contracted. Contracture is especially marked in cases where the hemorrhage breaks into the ventricle. When the face is contracted it is so drawn that the well side looks to be the weaker. Equal enlargement or contraction of the pupils is diagnostic of no particular condition, nor is divergence, since it may occur in normal sleep, but inequality is a sign that there has been cerebral mischief of some kind.

There is no reaction of degeneration in the muscles, nor any atrophy, except such as comes from disuse, nor is there fibrillation, since all such changes rest upon a degree of degeneration in the nuclei of cranial nerves, or the cells of the anterior horns of the cord, which in the present condition are intact, or only remotely affected, and to a slight degree, except in the rare instances where the nuclei in the pons or medulla are the site of the hemorrhage.

COURSE. An apoplexy has three critical points in time: 1st, death may occur in five minutes, and is quite likely to ensue in the attack, or within the first two or three days after it; 2d, after this the case may go on for a period of from two to four weeks, when it may take either one of two courses: (a) the patient may now die of exhaustion, or of some complication engendered by the paralysis; or (b) he may continue to improve until about the eighth week; when 3d, it is clear that he will make a relatively

complete recovery, or will remain in a state of chronic enfeeblement, which by this time has become pretty clearly outlined.

Starr sketches this as the course of an ordinary initial case. Some voluntary motion by the fourteenth to the twenty-first day. The first evidence is ability to draw up the leg, then to stretch it out, then next ability to move the ankle, and finally some flexion of the elbow and shoulder. The finer movements of the hand and foot are regained last of all, if they are ever regained.

PROGNOSIS. This is generally favorable for life in the first attack, but a recurrence is almost inevitable, and a third or fourth attack is rarely survived. A rapid and excessive rise in temperature, following the initial fall, is a sign of pneumonia, and an early fatality. A continued fall of temperature after the first few hours is of bad omen, as is difficulty of swallowing after several days have elapsed, or if consciousness is not regained in the first three days.

DIAGNOSIS. This is of two varieties; the first question is this: Is it cerebral apoplexy? and the second is, where is it located? The diagnosis of the fact depends upon a sudden attack of coma or complete unconsciousness, not dependent upon failure of the heart's action, followed by a hemiplegia. The only exceptions to this hemiplegic type are the extremely rare instances where the lesion is entirely sensory, or in the case of a primary localization in the ventricle, when the resulting paralysis is generally a double form of hemiplegia which is termed a diplegia.

Coma is a symptom common to many maladies, and therefore in all cases of coma obtain a sample of urine by catheterization, remembering that during the first 12 to 24 hours of an apoplectic attack the urine may, as a direct result, contain albumin, and sugar less often, and to a slight degree. Take the temperature in the rectum, note the respiration, state of the pupils, and of the superficial and deep reflexes. Examine the skull, and obtain all the possible data as to the manner of onset.

DIAGNOSIS OF LOCALITY.

Meningeal. This is either Dural, Subdural, or Subarachnoid.

Dural Hemorrhage. Dilatation of the pupil on that side is an important sign, and also unconsciousness, then a period of consciousness, and then unconsciousness. (Hutchinson's Pupil.) Pupil dilated on the side of the lesion from rupture of middle meningeal. This is in the Epidural, and is due to direct or indirect compression of the 3d nerve. It may come from the arteries, veins or sinuses of the dura, or the arteries of the pia. Subarachnoid attacks both sexes equally. The Subdural attacks three males to one female and is more frequent in youth and early adult life. The symptoms are those of pressure and paralysis; it is slow in onset, and of a rather limited area. Such paralyses are speedily relieved by operation, and do not tend to recur.

Cortical. This is likely to be focal and often multiple. It usually occurs after the membrane has been thickened by previous disease. Hemorrhages are generally small, but if large they extend into the subcortical white matter. The paralysis is likely to be a monoplegia, or of a limited area, since the cortical repre-

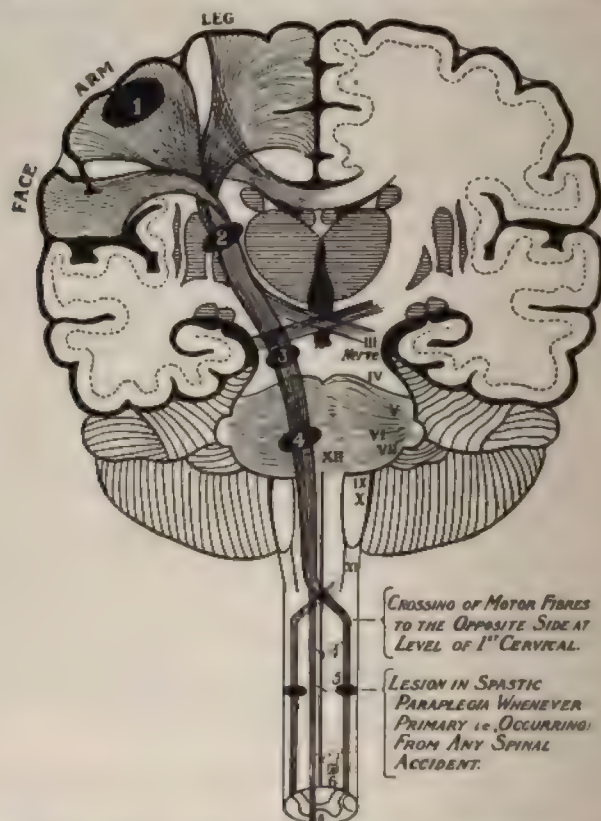


FIGURE 65.—LOCALIZATION OF THE DIFFERENT FORMS OF APOPLEXIES. IT WILL ALSO EXPLAIN THE SYMPTOMATOLOGY OF MANY TUMORS.

1. Cortical or Subcortical apoplexy; gives rise to monoplegia, or localized anesthesia, and convulsions. 2. Capsular apoplexy; give rise to hemiplegia or hemianesthesia, or both. 3. Hemorrhage into the Crus; this gives rise to a paralysis of arm, or leg, or a hemiplegia of the opposite side, and hemianesthesia, with rigidity and ataxia. It also causes a paralysis of the motor oculi on the same side as the lesion. 4. This represents a lesion of the lower third of the pons, and typically gives rise to a crossed or alternate hemiplegia; since it will almost necessarily cut off the fibres of the ascending sensory root of the fifth nerve, it will produce an anesthesia of the face on the side of the lesion, and on the opposite side of the body, since there is a sensory crossing in the lower part of the medulla. Since the lesion affects the fibres of the middle peduncle of the cerebellum, there will be rotation of the body to the side of the lesion. Any lesion between 4 and the level of the lower margin of the cerebellum as shown in the cut will produce this facial anesthesia, sparing the upper part of the face more and more completely as the site of the lesion becomes lower.

sentation of the whole of any one member covers a relatively large area. The hemorrhage gives rise to unilateral spasms, except in the rare event of a bilateral lesion. There is conjugate deviation of the eyes toward the site of the hemorrhage, a primary rise of temperature instead of the fall found in apoplexies of the brain-substance, and therefore is like that of thrombosis, embolism and hemorrhage into the pons and medulla.

Subcortical. A small hemorrhage just under the cortex will give rise to a group of symptoms which are partially irritative and partially destructive, and therefore, if it occurs in the motor zone, there will be the irritation symptom of a persistent spastic hemiplegia, or hemiparesis. If in the sensory area, we shall find exaggeration, or diminution of sensibility in the areas innervated from that locality, according as the lesion is destructive or simply irritative to the sensory cells.

Centrum Semi-ovale. This is rather frequent in the senile state, and gives rise to symptoms of irritation of the sensory, motor, or association tracts.

Capsulo-ganglionic. This is the typical cerebral hemorrhage and is marked by unconsciousness, hemiplegia, incomplete recovery and almost certain recurrence. If the rupture has occurred at the knee of the internal capsule, or at the anterior portion of the posterior limb we shall have a motor paralysis of the opposite half of the body. If the rupture is a little posterior to this, there will be a combined motor and sensory paralysis of the opposite half of the body, while if the posterior third only of this posterior limb is affected we shall have a paralysis of the opposite half of the body entirely sensory in character. This is very rare, but if present, we may have an ataxic form of hemiplegia without any actual motor paralysis.

Ventricular. The primary form is rare, while the secondary is much more common. Primary is caused by attempts at hanging, or by congestion from other causes which equally block the emissary veins. It may arise from concussion, and not be evident for days after the injury, or it may be the result of purpura, or pernicious anemia. The rupture may be of the choroid-plexus, velum-interpositum, or a vein of the wall of the ventricle, and from such causes as would naturally be expected to provoke one of the more common types of cerebral apoplexy. It may occur in the young, even in infants at birth. The symptoms are stupor, sluggishness, paraplegia, or quadriplegia, and a liability to general spasms without any preceding stage of unconsciousness; spasms following unconsciousness would lead one to suspect a hemorrhage in or on the cortex. A small hemorrhage confining itself to one ventricle may cause very slight symptoms, but if it invades all the ventricles, as is the usual rule, we shall find not unconsciousness, but dullness, paraplegia or quadriplegia, accompanied

by rigidity, and, at times, by unilateral spasms. If the force of the hemorrhage is great, or the quantity large, it will invade the cavity of the fourth ventricle, or violently push before it the fluid normally filling the ventricular cavities, and so injure the nuclei of the cranial nerves originating there that it generally causes death. An alteration of the respiration and the action of the heart is a sign of such invasion, and this is the result in one-third of these cases. Secondary ventricular hemorrhage results from the extension of a hemorrhage originating in the capsule, or its neighborhood, through the yielding tissues into the cavity of the ventricle. Such cases fail to come out of the initial state of coma, but when a day or two has elapsed after the primary apoplexy, the coma deepens, the temperature rises to 103 or more, the respiration becomes Cheyne-Stokes in rhythm, the pulse grows tumultuous, and the patient dies. This has often been wrongly interpreted as Ingravescens Apoplexy, which is a condition where an apoplexy, normal in its after history, takes from a few hours to a day or two to reach its maximum of intensity.

Cerebellar. This is located in a large proportion of the cases in the vermis, next, in order of frequency, in the lateral lobes near the corpus denticulatum, and less often in the medi-peduncles. The diagnostic feature of such a hemorrhage is a sudden paralysis, usually without unconsciousness, but characterized by vomiting, and intense vertigo and ataxia. This vertigo is intensified by standing or sitting, and is termed *Astasia-Abasia* (inability to walk or stand). If the hemorrhage is in or near the medi-peduncle, it will give rise to forced movements of the limbs, and a rigid position of them and of the body, which may cause it to assume positions of flexion in any direction, which will be retained even while lying in bed. There is immediate ataxia, and weakness of the extremities. Tetanoid spasms are present at times, while sensory symptoms may be absent or transient. Any hemorrhage produces a mass which, in relation to the structures near it, may be regarded as a tumor, and therefore, while the paralysis produced primarily by a cerebellar hemorrhage usually is upon the same side as the lesion, the secondary effects of pressure upon surrounding structures may complicate the picture. If the pyramidal tracts are compressed by it, above the point of their decussation at the level of the first cervical vertebra, we shall have a motor paralysis of the other side of the body, while some of the cranial nerves may show paralysis of the same side as that of the hemorrhage. If, on the other hand, the pressure is exerted at a point below their decussation, the paralysis of the body will be upon the same side as the hemorrhage. Motor paralysis does not always result. Its absence, and the presence of the symptoms of spasm and ataxia noted above, have caused cases of this variety to be considered hysterical. The pressure on the cranial nerves

and descending motor tract may be only sufficient to give rise to symptoms of irritation, but, since the tissues of the cerebellum are very soft, and the velum between it and the floor of the fourth ventricle is extremely delicate, it is not unusual to have a rupture into the cavity of the fourth ventricle. In such a case we shall find symptoms as detailed above. There will be a paralysis of the lower cranial nerves, and especially alarming symptoms from the injury to the pneumogastric, while the vertigo will be increased and will be labyrinthine in type, and only distinguished from it by a history of freedom from previous aural disease. The diagnosis may be further complicated by actual injury occurring to the nucleus of the eighth nerve.

Pontile. The hemorrhage is usually from the median branch of the basilar artery, and the raphe is so firm that it is confined to one side, and a hemiplegia is the usual result, but the blood may be from the transverse artery, and in such case primarily attacks the nucleus of the fifth nerve. It may extend into the middle peduncle of the cerebellum, or the fourth ventricle. Consciousness is not always lost; there may be initial convulsions of a general character, but, in rare instances, of one side only, and sometimes affecting only the legs, which latter localization is diagnostic of pontile hemorrhage. There may be a marked loss of sensation on the opposite side of the body, and this may be the preponderant symptom. The peculiarities of this form of apoplexy will be more readily comprehended, if we recall that the pons is transversed vertically in its anterior portion by the pyramidal fibres descending to all the body, and the root-fibres of the 5th, 6th and 7th cranial nerves. The decussation of the 7th, or facial nerve fibres, takes place just at the level of the apparent nucleus of origin of the 5th nerve. If then, a hemorrhage should destroy the facial fibres before they decussate, and at the same time cut off the descending pyramidal fibres, we should have a paralysis of the face and of the limbs on the same side, which would be that opposite to the site of the clot. If, however, the facial fibres had already crossed, the decussation of the motor fibres to the body being far below this point, we should have the face paralyzed on the same side as the lesion, while the paralysis of the limbs would be on the opposite side. This is a typical "Crossed" or "Alternate" hemiplegia. The sensory fibres in the pons lie above and behind the motor fibres in the area called the "Tegmentum." If the lesion is deep enough, it will cause a sensory paralysis on the side opposite the lesion. If the motor nucleus, or root-fibres of the 6th, or abducens are injured, the movement of the eyes toward the lesion is impossible, since the fibres of this nerve are peculiar in having a double decussation, or, as some believe, in not decussating at all. The motor and sensory portions of the 5th are close together, and may be affected together, or separately, and

if the motor-nuclei are irritated, but not destroyed, trismus may result. The sensory and motor losses may be crossed, and the face may be insensitive upon one side, while the paralysis of the limbs is on the other. Sensory losses are frequent, and may predominate. Absence of a hemiplegia is more significant than its presence. There are early rigidity, facial paralysis well-marked on the side opposite to the paralysis of the body, and a conjugate deviation of the eyes away from the lesion. If the lesion is irritative only, they look toward the lesion. There is great liability to spasms and to early rigidity, which is late in other localizations. The pupils are pin-point, instead of dilated, as in cerebral lesions. Initial rise of temperature occurs, which goes on to hyperpyrexia, as in thrombosis and embolism, and the exact opposite of the initial fall of cerebral lesions.

Crus. Hemorrhages in this locality may be downward from the thalamus, or corpus striatum, or may be primary, in which latter case they are small. If diffuse they are quickly fatal, but if circumscribed, and in the ventral portion of the crus, they destroy the motor tracts to the whole of the opposite side of the body. The lower face is also paralyzed, while, by destruction of the 3d nerve-nucleus, there is paralysis of all the muscles of the eyeball, except the superior oblique and the external rectus, on the side of the lesion. The 3d nerve issues from the inner side of the crus. Crossed hemiplegia with 3d nerve implication is the "Benedickt-Weber" syndrome. Headache is trifling, optic neuritis is unknown, convulsions are rare, and mental changes are slight, and less than in softening. Sensibility is blunted, but hemianesthesia is rare. Increase of sensibility to pain may be present, and co-exist with a decrease of ordinary sensibility. There may be spontaneous pains, but less often than in softening. The hemorrhage may go up into the foot of the capsule, and then we shall find hemianesthesia.

Medulla. On pathological grounds we should expect a hemiplegia of the limbs and trunk on the opposite, and of the parts innervated by the lower cranial nerves on the same side as the lesion, but the area is so small, and the nuclei govern such vital processes, that a lesion here usually means death.

The diagnostic mark of all hemorrhages of the crus, pons and medulla is a crossed paralysis. This means that the paralysis of the body is on the side *opposite* to the lesion, and of the cranial nerves, on the *same* side as the lesion. The level of the lesion will be indicated by the particular nerves implicated. Lesions of the crus *directly* affect the 3d; of the pons the 5th, 6th, 7th, and possibly the 8th, while those of the medulla affect the 9th, 10th, 11th and 12th.

DIFFERENTIAL DIAGNOSIS. Syncope. This and the three next conditions are barred out if there is a definite one-sided paralysis.

In syncope we find a pale face, sighing respiration, consciousness only partially abolished, dilatation of the pupils, preservation of the deep reflexes, and rapid recovery.

Narcotic Poisons. *General* convulsions never occur. Opium produces a slow respiration, small and thready pulse, cool and blue skin, and a most extreme contraction of the pupils, and the corneal reflex is never lost. Temperature is low, there is no paralysis, and coma is never absolute.

Alcoholic Intoxication. *General* convulsions never occur. There is the odor of alcohol, nature of the vomit is diagnostic, there are motor restlessness, tendency to delirium, absence of a low or unequal temperature. Remember that a man may become intoxicated and rupture a weakened artery, while he is also more liable than a sober man to fracture his skull. In such cases, his being alcoholic does not bar out apoplexy. There is no paralysis, nor are the pupils unequal.

Test for Alcohol. Add a few drops of urine to the following mixture: Solution of Potassium Bichromate, 1 part; Sulphuric acid, fortior, 300 parts. The result will be a bright emerald color. (Turner in Allehin).

Uremic Coma. The condition cannot be certainly diagnosed, while the patient is in coma, by the urine alone, since apoplexy tends to produce an albuminous urine for the first 12 to 24 hours, although there are no casts in the urine of an apoplectic *from the apoplexy*; if present they are from a pre-existent nephritis. If there are convulsions present which tend to shift about, they are probably uremic, while if unilateral the cause is probably cerebral apoplexy. The history of previous nephritis would be a guide. Uremic coma comes *after* vomiting, headache and convulsions, while the vomit of apoplexy comes after the coma, and the coma of uremia is not absolute, and the patient is restless, instead of lying like the apoplectic in perfect apathy.

Meningeal Hemorrhage. This is like cerebral hemorrhage in the presence of stupor, coma, and convulsions, but if there is a history, it is one of injury, and there has also been an interval between it and the coma of hours, or even several days.

Epileptic Coma. An epileptic attack presents no particular similarity, since there has been a tonic, followed by a series of clonic convulsions, followed by stupor, while the reverse is true in the convulsions of apoplexy; but if this history is unknown, and we find a person in the stuporous stage, which may be unusually prolonged, or replacing the usual attack, we should then examine the tongue for bites, or scars of old bites. Unilateral symptoms are rare, the patient is youthful, arteries are good, there are capillary hemorrhages in the conjunctiva, and on the cheeks. The knee-jerks are abolished in both conditions; exaltation of the deep reflexes may be found for a few moments in both. Apoplexy

has been known to occur from the cerebral strain of an epileptic convulsion, and therefore is a possible complication.

Focal Epilepsy. This may indicate a cortical hemorrhage, and is common in all diseases of the motor cortex. The attack may resemble an apoplectic attack, and the history alone is a guide, since the paralysis may exactly resemble an apoplexy. In a young person think of multiple sclerosis.

Hysteria. At times it looks very like, but the patient is apt to be young, the face is exempt from paralysis, and betrays the presence of hallucinations. Stigmata of hysteria may be present, and psychic suggestion, or ovarian pressure, may terminate the condition at once. The pulse may be *accelerated*, but is never *retarded*; the pupillary reflex remains, and the deep reflexes also, and there is no Babinski. Temperature is normal, paralysis is flaccid, and there is often anesthesia.

General Paresis. The condition in this disease which simulates apoplexy is a true apoplexy, in the sense that it is a dural hematoma from the rupture of an artery in the meninges. It is much sooner and much more completely recovered from, as the brain is not torn, but only compressed. The knee-jerk is exaggerated, there are facial tremor, speech defect, Argyll-Robertson pupil, and a history of grandiose, or melancholy delusions. There is a history of mental failure.

Diabetes. There is a uriniferous (and sweet) odor of the breath found in no other complaint. The coma is not of sudden onset, but is the culmination of a condition of somnolence and headache, anxiety, and sensations of suffocation of some duration. Urinary examination determines the diagnosis more than in uremic coma, since the glycosuria of apoplexy is very slight.

Fracture of the Skull. In almost all cases there is bleeding from nose, mouth or ears. The coma in these cases is due to the rupture of a meningeal vessel.

Acute Softening from Embolism and Thrombosis. The embolic patient is young, valvular disease is evident, or there is the parturient condition. Coma is shorter or slighter, and the temperature is only slightly disturbed, and if so is elevated, and not depressed, and paralysis comes first and then coma comes on after convulsive movements, which is the reverse of the order in apoplexy. The face is pale, the pulse is small, and the onset is generally gradual, but may be as sudden as apoplexy. There is no unilateral variation of surface temperature.

Thrombosis. The patient is old; or if young has had syphilis; or if an infant has had marasmus, or some similar disease. The onset is often during sleep, and for some time previous there has been some blunting of intellect, confusion of ideas, and of intelligence. Restlessness at night is a very common prodrome. The

pulse is irregular, and we find hard radial arteries and there is a condition of general atheroma. The pupils are even, and react to light; the face is pale. Coma is light, and twitchings show which side of the body is to be affected. Twitchings are common, while convulsions are rare. Coma lasts only for from twelve to twenty-four hours. No *fall* of rectal temperature. Thrombosis usually occurs in sleep, and when blood-pressure is lowered; while apoplexy is the accident following an elevation of blood-pressure. The explanation of the fact that many apoplexies occur in sleep is that the venous return is impeded by position, or by some compression of the veins.

TREATMENT. It is rare that a meningeal apoplexy can be diagnosed with certainty. I have seen one case where the localization was evident, and the clot was removed. Starr quotes a case from McBurney where the clot was found, and removed, and the patient recovered. The result was not so favorable in my own patient. When the hemorrhage is cerebral, we are combating bleeding from a vessel which is inaccessible. Can we check such a hemorrhage? The general opinion is not favorable to the success of such measures. Some of them do no harm, nor do they add to the discomfort of the patient, and they should be employed. The ice-cap has been employed to counteract dilatation, and to contract the arteries of the brain. Swathing the limbs in hot bandages attracts the blood to those parts, and reduces the blood pressure in the brain. Sinapisms are applied to the calves and the soles of the feet, and for the same reason they are applied to the nape of the neck for the purpose of directly contracting the arteries of the brain through vasomotor stimulation. Norwood's tincture of *Veratrum viride* is given in doses of 2 drops every 20 minutes, until the pulse is soft. This is used for the purpose of so relaxing the arterial tone that the patient bleeds into his own veins. Means of a much more drastic kind have been proposed. Bleeding, 12 ounces. Ligation of the common carotids, based upon the statement of Spencer and Horsely that such a procedure in monkeys produced a fall in the cerebral pressure. Compression of the carotids for forty minutes, if the case is seen early, is suggested by Church. Schwartz has advised arteriotomy of the anterior branch of the temporal, on both sides if necessary. Dawbarn and Thompson have advocated the use of the Spanish windlass on the limbs, close to the body, for one hour, just tightly enough to obstruct the venous return of the blood to the body. All advise clearing out the intestinal tract, if it has not been done spontaneously, and the medicament which does this, with the least disturbance of the patient, is 2 drops of Croton Oil in a little butter, which is to be placed upon the tongue.

With all these recommendations before us, it seems still that the best plan is to let the patient severely alone until a later

stage. We should avoid shock most scrupulously in moving the patient, and, if the clothing is difficult to remove, it should be cut from the body and the neck-bands should be loosened in every case. Whether, or not, position reduces blood-pressure, it seems wise to place the person on the paralyzed side, since the saliva thus runs more easily out of the mouth. Keep the mouth closed by favorable position as far as possible, since the drying of the mouth is thus mitigated. Do not feed, since the patient can neither swallow, nor digest, and administer water by wetting the mouth and the lips. Keep the room cool, dark, and quiet, and do not allow the solicitude of the friends to annoy the patient. When consciousness returns, administer plain and unstimulating food in small quantities, but never *raw* milk. There should be no stimulants, but fluids of a refreshing character in moderate amounts. Easily digestible solid food may be given at the end of a week. The patient should have his position changed frequently, and he should be sponged with alcohol and water, but in all these measures he should be warned against effort on his part. These changes and cleanliness, and keeping the skin dry, is the best method of combatting bedsores. The bladder should be emptied by a catheter, and the bowels should be kept open.

Drugs have a limited sphere in the treatment of this accident at any stage, and in the later ones massage and faradic electricity should be faithfully used to develope to the full the wasted muscles. Contractures are benefitted by them and the patient should be encouraged to attempt movements which are imperfectly accomplished. Contractures of the hands should be antagonized by gripping motions, and the weak ankles should be moved to and fro against resistance. Muscle-beaters can be used with advantage on the contracted muscles, and warm baths followed by oil-rubs will be found helpful.

Electrical Treatment. In predicting the value of electricity in a given case of apoplexy, it must be remembered that this agent will not restore nerve-cells which have been destroyed, nor will it replace nerve-fibres whose nutritional source was destroyed when the cell died. It will stimulate cells which were injured, but are still capable of function, it will preserve the volume and integrity of muscles which are only awaiting the recovery of the part of the cortex from which they are innervated. Its value in accelerating the absorption of a clot is rather doubtful. If it can affect the clot favorably, it will do this under the influence of a galvanic current passed through the skull so that the clot lies in the field of force. If it does produce any effect, it will be manifest within three weeks and it should not be employed until three or four weeks have passed from the attack. The anode should be placed over the lesion, and the cathode at various points on the opposite side of the head. A more powerful method is to place both electrodes

upon the same side, but the anode should be kept over the lesion. General galvanization to the spine will increase the nutrition of the spine, and secondarily of the whole body. At another sitting, so as to avoid fatiguing the patient, the muscles should be exercised by placing the anode upon the back, and the cathode over the motor points of each affected muscle with an interrupted current. This fatigue is shown by a decreased motor response to the current. Contractures are due to degeneration of the descending tracts in the cord, and are incapable of much benefit. The proper treatment is outlined in the article upon Spastic Paraplegia. Aphasia is generally due to the destruction of the cells in some one of the speech centers, and is not amenable to treatment. If the cause is an ischemia, or the temporary shock to the cerebral circulation, it will be benefitted by placing the anode over the posterior part of the third frontal convolution, and the other on the right side of the larynx, passing a galvanic current for two or three minutes. Then place the anode on the back of the neck, and the cathode, using a small Erb's electrode, on the motor-points of the external and inferior laryngeal nerves, pressing the electrode firmly into the tissues. The static breeze is valuable as general nervous stimulant, and strong sparks over the dorsal spine seem to improve locomotion, and over the cervical plexus are beneficial to the movements of the arms. Anesthesias are also relieved by the static breeze, and the faradic brush. The galvanic anode relieves pain in the muscles at times.

THERAPEUTICS. If we should chance upon one of those rare cases where there are premonitory signs of confusion, Glonoin in doses of 1-100th of a grain may be given, or Aconite, or Belladonna in potency, or Veratrum viride in doses of a drop of the tincture every hour until the pulse is softer. If excessive headache is the prominent symptom, Melilotus is valuable.

During the attack we can use our remedies with advantage if the dose is a few drops on the tongue. Avoid even spoonful doses. In the stage of onset, Aconite is as beneficial as bleeding, but if there is deep coma, use Opium; if the person is sthenic, Belladonna is preferable, and if the hardness of the pulse is conspicuous, Veratrum viride as suggested above. Where the heart's action is irregular and feeble, use Glonoin in the first dilution. Where the condition is apathetic, as though hemorrhage were continuous, the use of Hamamelis in tincture is advisable.

When the stage of irritation has come on, Bryonia in the lower potencies is of value, but Belladonna, Apis, Hellebore, or Cicuta may be more specifically indicated. If the only symptom of this is headache, indicating implication of the meninges, we should then consider Bryonia still the better remedy if there is great tenderness of the scalp, but if this is not the fact Melilotus is preferable. About the eighth day the movements of the limbs indi-

cate that compression is reduced, and Ignatia, or Nux vomica 3x are of service.

In the later stages, after the patient's condition has become a chronic one, Nux vomica in the lower dilutions seems to accelerate recovery more than any other drug, but if the case comes to a halt under its use, Baryta carb., Causticum or Hepar may be found of use. Strychnia phos. 3x, or in material doses, or Zinc phos. 2x, will accelerate improvement.

SOFTENING OF THE BRAIN.

CEREBRAL THROMBOSIS AND EMBOLISM.

DEFINITION. Although this condition is an end-product of a number of different processes, its consideration as a definite symptom-complex has been sanctified by long usage, and there is no reason for its abolition. It is that condition of necrosis of the brain-substance which results from capillary hemorrhage, or from the occlusion of larger vessels by thrombosis, or embolism.

SYMPTOMS OF A TYPICAL CASE. These must be separately described. If it is the result of capillary hemorrhages, unconsciousness will be rare, but the intellect will be clouded, and there will be bodily restlessness. Single muscles will be paralyzed, or single members, and the permanent loss will be a weakness and clumsiness of movement. If the cause is thrombosis, the patient will suffer for a time with insomnia and headache, and a confusion of mind; some grade of paralysis will come on, probably without loss of consciousness, and this paralysis will be widespread at first, but will be quickly reduced to much narrower limits. Aphasia is a common result. If embolism is the cause, the attack will closely resemble an apoplexy, but the paralysis will soon pass away with comparatively slight remnants.

DIFFERENTIAL DIAGNOSIS. It is to be diagnosed from massive Hemorrhage, Hematoma of the Dura, Tumor and Abscess of the Brain, and Encephalitis.

AGE. It is a disease of adults, and if it is Embolism the patient is generally in the full vigor of life, but in Capillary Hemorrhage, and Thrombosis, the patient may be very young, but is generally old. Syphilis may cause this lesion at any stage of life.

ETIOLOGY. Any disease or cachexia which degenerates the coats of the arteries will render the individual liable to this accident, and we are therefore likely to find it in syphilis, the later stages of nephritis, diabetes, alcoholism, and intoxications by the poisonous metals. The peculiar changes giving rise to Paresis, Progressive Bulbar Palsy, and Multiple Sclerosis entail this probability, and it frequently occurs from the arterial degeneration con-

sequent upon old age. Thrombosis is the formation of a plug in an artery, which is formed at the point where it is found. It is due to changes in the endothelium of the arteries, and is therefore liable to be found in alcoholism, syphilis, lead-poisoning, and gout. Atheroma and arterio-sclerosis roughen the intima, and in their degeneration so weaken the arteries that they easily rupture under normal pressure. In their earlier stages the roughness needs only the added element of an increase in the coagulability of the blood, or a slowing of the circulation, or both, to invite the formation of a thrombus. Old age and the debilitating diseases of childhood furnish all of these elements, and so this accident is common in the sleep of the aged. Phthisis and the puerperium both tend to produce some degree of endarteritis, and the blood is also more coagulable under such conditions; hence the accident is not uncommon under these circumstances.

An Embolus may arise from particles of tissue carried to that point from vegetations on the valves of the heart, resulting from rheumatism, typhoid fever, pneumonia, diphtheria, and the puerperal state. It occurs also after lacerating wounds, and from the degenerating areas in carcinomata, and other like processes. After extensive burns, fat emboli often occur. As a clinical fact, however, it practically always results from some previous cardiac condition which has produced vegetations about the mitral orifice, and which have been dislodged by some sudden exertion, or sudden call upon the contractile power of the heart.

PATHOLOGY. If a plug forms in a small terminal artery of the cortex, we shall find a small bloodless area, surrounded by a larger one which is congested. There is an exudation of serum, and an invasion of leucocytes by the third day. The prevailing color is at first white, but this soon becomes red, on account of the reduction of the serum, and the presence of the extravasated blood corpuscles. The leucocytes are now filling themselves with the disintegrated blood-cells, and the debris of the tissues, and the parts become yellow, which condition is reached in about six weeks. In about sixty days, the leucocytes will have cleared the area of all dead tissue. When, therefore, we speak of white, red, or yellow softening, we are speaking of different stages of a single process. If the spot was a very small one, there will be a minute mass of neuroglia tissue (a scar), but if it is larger it will be a cavity, or a cyst, filled with clear fluid. The part is dead, functionally speaking, since nerve-cells die in about forty-eight hours, if deprived of their blood-supply. In the case of a thrombus, or embolus, we find, instead of a more or less circular spot, an infarct, in which the subsequent process is the same as has been just described. Thrombi tend to form in the large central arteries, while emboli affect those of smaller calibre. They also are generally situated at the bifurcation of arteries, and may grow

backward by accretion from the blood-stream, or may subsequently become perforated, so that the area again is nourished to some proportion of its former supply. An embolism not infrequently follows the disintegration of a thrombus, and thrombosis may succeed embolism. If the embolism occurs from a fragment from the site of an infection, as ulcerative endocarditis for instance, we shall find that the infaret is infected, and an encephalitis will be the result. The clinical results differ according to the size of the occluded arteries. If a large artery is closed, a large area of the brain is affected, and the initial shock is great, but from

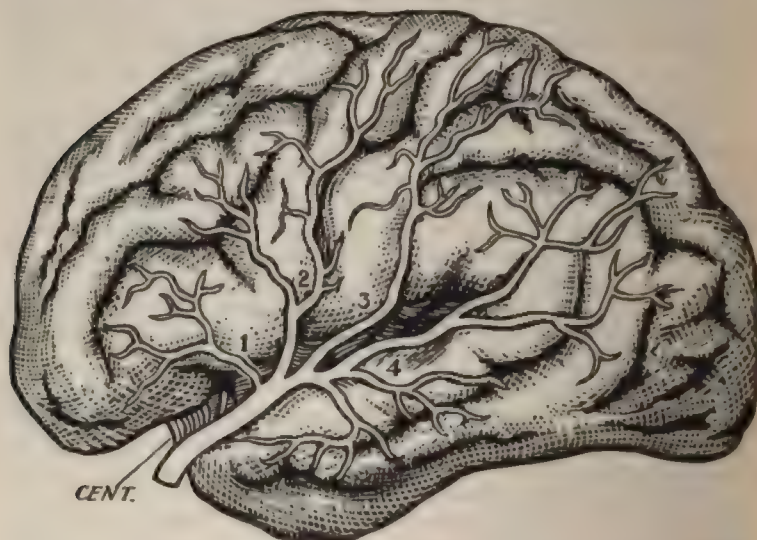


FIGURE 66.—THE DISTRIBUTION OF THE MIDDLE CEREBRAL ARTERY.

1. Subfrontal branch. 2. Precentral. 3. Postcentral. 4. Parieto-temporal and Temporal. Cent. Central branches. (After Duret.)

anastomosis a new avenue of supply is soon developed, and the paralyses will be transient. The small arteries are terminal, and there is no anastomosis; therefore, while the initial shock is quite slight, the resulting paralysis, such as it is, will be permanent.

Localization is quite fixed in relative frequency. It is much more common on the left side of the brain; capsulo-ganglionic. Next we find it in the cortical distribution of the middle cerebral, next the posterior, and lastly of the anterior cerebral artery. It is a rare accident in the cerebellum, or in the bulb, except in the syphilitic. It is never bilateral, except when it occludes the circle of Willis.

SYMPTOMATOLOGY. When small capillary hemorrhages occur the patient will show a variable degree of shock, proportionate to the size of the vessel or of the number simultaneously ruptured, but in any case, the symptoms are not general shock to unconsciousness, but rather a clouding of the faculties, and a paresis of single parts, or even of single muscles. This loss is usually permanent as a simple weakness or clumsiness, since the cortical area of innervation of a single movement is distributed over a considerable area.

Thrombosis differs from both multiple hemorrhage and embolism in having a premonitory stage, which may be days, or even weeks, in duration. The patient is subject to headache, and reduced mental ability, and, at times, irritability. The patient is apt to be petulant, and is constantly seeking some cause of his unrest in his environment. A patient of my own, under the influence of a forming thrombus, changed his room in a hotel four times on one night, and had his bed made and re-made a half-dozen times. The muscles are generally weakened, and sleep is greatly disturbed. When occlusion is completed, we shall find one of two classes of symptoms. If the occluded artery is large the patient will be found, generally after sleep, to be clouded in mentality but, while difficult to arouse, is not unconscious; coma and unconsciousness may be as marked as in apoplexy. He will be paretic, meaning by that, weakened muscularly and incoordinate, but not actually paralyzed, and often aphasic. This condition will endure for a few days, when it will improve, and the aphasia generally shows improvement, and in a week or two disappears except that some degree of hesitation in speech remains. When the patient is tired, or emotionally overtaxed, he is liable to a temporary renewal of the aphasia. Initially there is a rise of temperature of one or two degrees, and the pulse is rapid and small. If the thrombi are multiple, and small and widespread, a common form in the aged, there will be no initial shock, but the result will be an increasing feebleness, accented on the days when a new vessel is occluded. The patient is progressively more and more feeble in mind and body, the muscles lose their tone, and there is a marked incoördination of movement, so that the patient's movements are clumsy and weak.

Embolism differs from the two preceding causes of softening in the fact that it is as abrupt as massive cerebral hemorrhage, and has none of the premonitory symptoms so usual in thrombosis. There may be all stages of mental confusion, or coma, and complete unconsciousness; there may be spasms, or epileptiform convulsions; mental confusional states may alternate with delirium. Vomiting is quite frequent. The temperature is elevated, as in thrombosis, and the pulse, here also, is weak and rapid.

In both thrombosis and embolism, the patient is apt to suffer

extremely from muscular cramps, and all sorts of paresthesias. There will be stinging pains in muscles and skin, and burning and other unpleasant sensations, which may be so severe as to prevent sleep. While the paralysis in both conditions is initially widespread it will improve far more than we should expect, and much more rapidly, yet it must be remembered that it is not a negligible condition, and that which persists more than three weeks is liable to represent a permanent loss. The muscles lose their tone from atrophic conditions, and this is the condition in all sorts of tissues, and may be severe enough in grade to produce bedsores. The muscles of respiration, and of the heart may be affected, and their implication indicates extreme danger to life.

PROGNOSIS. Minute, multiple hemorrhage is bound to be recurrent, and finally will produce death, because the conditions are such that no regeneration of tissue is possible, and because the small scars or cysts are foci of continuing cerebral irritation. The same probability of recurrence is true of thrombosis, but embolism is not nearly so liable to recur, and the age of the patients gives greater promise of recuperation. Many cases of embolism die in the attack, as do some cases of thrombosis, but multiple hemorrhage almost universally is the initial step in a gradual physical and mental deterioration, lasting for some years. If the case of embolism revives after a day or two, the prognosis for life is good, but in thrombosis there is a second period of acute danger, at about the tenth day, from secondary softening. Bulbar symptoms indicate a probably fatal issue.

DIAGNOSIS. This rests upon general and special features. Generally upon a condition of widespread muscular paralysis, which is soon diminished to an enfeeblement, mental loss of all grades, from confusion to unconsciousness, with a rise of temperature, a weak and small pulse, and a pale face. Specially, between the different causative conditions, as has just been detailed. Brain symptoms, accompanied by a bloody urine and pain in the lumbar region and an enlargement of the spleen, denote infarcts in those localities, and indicate that the brain condition rests also upon cerebral embolism.

DIFFERENTIAL DIAGNOSIS. Massive Hemorrhage. This differentiation is not always possible, but it is almost certainly hemorrhage if we find deep coma, a retarded, strong, and full pulse, redness of the face, and the temperature of the paralyzed side of the body elevated.

Hematoma of the Dura. (*Hemorrhagic Internal Pachymeningitis*). This is generally bilateral, but not equal, on both sides of the body. Marked by headache, vertigo, a tendency to somnolence, and some grade of paresis. More often affecting the limbs than the face. The pupil on one side is apt to be contracted. Spastic contraction of the limbs, and conjugate deviation of the

eyes, sometimes. Occasional convulsions, which may be unilateral, or general. Intervals of health, somewhat disturbed by brain-symptoms. Very chronic. Generally preceded by an accident, or precursory pressure-symptoms.

Tumor. Marked headache, convulsions, vomiting, choked disc with no paresis or mental reduction.

Encephalitis may arise from an infected thrombus, but if primary, there is the history or presence of a focus of infection. Somnolence is marked, there are rapid rise of temperature, and convulsions ending in death.

Abscess. There is the history of an infective injury, or purulent disease of the ear, or nose, or the antecedent presence of an embolus from some infected area, especially in the lungs. There are general cerebral symptoms, with a subnormal temperature, or fever and chills.

TREATMENT. This should be like that of apoplexy, modified by the evident difference of the conditions.

SINUS THROMBOSIS.

DEFINITION. It is an occlusion of the sinuses of the cranium by a coagulum, primary or secondary.

ANATOMICAL CONSIDERATIONS. The transverse sinuses, and the inferior petrosal, empty into the internal jugular vein, as well as all those centering at the Torcular Herophili. The external cranial veins, the auricular, and cervical also, form the external jugular.

The internal nasal veins communicate with the superior longitudinal sinus. The external cranial veins into the transverse sinus through the parietal foramen. The ophthalmic veins run into the anterior portion of the cavernous sinus. The veins of the middle ear empty into the lateral sinus.

ETIOLOGY. Primary thrombosis is due to a combination of the reduction of the rate of the blood current, and an increase in the coagulability of the blood. It arises also from a decrease in the quantity of the blood-mass, but entails one other condition, since it is not probable that a decrease in an actual quantity would be unaccompanied by some change in its consistency. It is an incident of Infancy when exhausting disease is present, and is then called Marantic. Owing to the stage of life the heart does not retain its power, and the circulation is slower than normal, and an exhausting diarrhea has at the same time increased the coagulability of the blood. Old age presents the same favoring conditions, and, in addition, there is generally some degree of atheroma of all the blood-vessels. Exhausting diseases, and the cachexiae may, as an unusual result, from a weakened heart, coagulable

blood, and a degenerated endothelium, produce this condition in persons of any age. Chlorosis has caused it. The usual site is the superior longitudinal, while it is rare in the lateral, and very uncommon in the cavernous sinus. There may be a primary infection of a sinus, or thrombosis may arise from compression.

Secondary Thrombosis. Infections in the vicinity of a sinus may induce a phlebitis of the sinus, or it may be an extension from a similar process in a tributary vein. The most common source is a suppurative process in the middle-ear, or in the bony cells communicating with it. The lateral sinus is the one affected by such a process. Infected wounds, or abscesses of the scalp, may infect any of the sinuses lying near the margin of the skull, the superior longitudinal being most often the one implicated. From similar processes in the orbit, nose, and face, the inferior longitudinal, or inferior petrosal, or cavernous become the site of thrombosis.

PATHOLOGY. The vulnerability of the sinuses rests upon the fact that their lumen is crossed by numerous trabeculae, Pacchionian bodies project into them as the years increase, veins empty into them against the direction of the blood-current, and the direction of flow is often against gravity. In primary cases the coagulum is at first red and soft, while in later stages it is whiter and harder. In infections and secondary cases it is varied in color, with collections of pus, and abscesses may form in the wall in the vicinity. Thrombosis of one sinus may extend to the analogous one on the other side of the cranium, or for some distance into the contributing or emissary veins. In any case the contributory veins of the cranium and brain are congested and tortuous, and that part of the brain and meninges is in a state of congestion. This may give rise to capillary hemorrhages, infarcts, areas of softening, and abscesses. The blocking of venous drainage may induce hydrocephalus.

SYMPTOMATOLOGY. The causative conditions generally furnish most of the symptoms, and may have been previously so severe that the new condition adds few new elements to the symptom-complex. The general symptoms, when noticeable, are these: In primary cases the following symptoms may be present: There are prostration, severe headache, vomiting and stupor; then delirium with unilateral, or generalized convulsions, and paralyses, and, more rarely, incoördination, contracture, tremor, etc. There may be rigidity, nuchal and general; hemiplegia, paralyses of the cranial nerves, especially ocular. The temperature is normal or elevated to 108, going up by bounds; pulse normal or retarded, rising later; respiration similar. These symptoms are like a meningitis with softening.

Secondary: A thrombosis of the lateral sinus, which is not septic, and is discrete may give rise to no symptoms beyond headache and fever. Generally the symptoms are characteristic, be-

cause an abscess outside of the sinus and the dura is generally the transmitting medium. The symptoms are inflammation of the bones, sub-periosteal abscess, phlegmon behind the mastoid process, especially around the mastoid foramen, and the neighboring part of the occiput, also on the mastoid process itself. We find pain on pressure and percussion of this region, limitation of movements of the head, then the general symptoms mentioned in the primary cases, plus optic neuritis, and choked disc.

LOCALIZING DIAGNOSIS. Sinuses in front of the skull, if occluded, produce dilated frontal veins, cyanosis of frontal and orbital region, swelling of eyelids, protrusion of the eyeball, maybe blindness.

Transverse Sinus; swelling behind the ear, and of the veins of the skin, but the jugular is also involved in most cases.

Cavernous Thrombosis may be suspected, if, after an attack which gives ophthalmic vein symptoms, we get an extension to the other side.

Occulsion of the Lateral Sinus involves the jugular vein most often. Systemic symptoms, chills, fever even to 108° with stiff neck, vertigo, vomiting, retarded pulse, irritability, sensitiveness of spinous processes, all meningeal symptoms. Symptoms in the lower extremities have also been observed, Westphal's sign, contractures, paraparesis, etc.

* The Symptom of Thrombosis in children, to diagnose between it and the Hydrocephaloid of Marshall Hall, is that the fontanelle is elevated, while in hydrocephaloid it is depressed.

Marantic almost always in the posterior part of the longitudinal sinuses, and may also be found in the transverse sinuses.

Otitic. Mostly in the transverse at the sigmoid fossa; at times in the cavernous, and the inferior and superior petrosal.

Traumatic in the superficial sinuses. Injuries to eyeball, face, and nose in the cavernous.

TREATMENT. In the cases where it affects the lateral sinuses surgical treatment is advisable, and is often curative. It is rarely of any avail in the longitudinal sinuses, and the internal are beyond our reach. In primary cases the patient should be kept in a recumbent position, and, if in the longitudinal, the neck should not be flexed. In the marantic cases the causative condition engages all our attention, but in any case ice-bags on the head will be needed. Attention should be given to the eliminating functions of the bowels, kidneys, and skin, and a supporting diet with stimulants should be administered.

THERAPEUTICS. There are slight grounds for much hope from the action of remedies, but Lachesis, Naja, Crotalus and Arsenicum have been recommended. The basis for their use rests upon the clinical fact that recoveries have taken place in the experience of the author which seemed to have some probable relation to the

action of the remedy. It should be remembered that spontaneous recovery is not impossible.

THE CEREBRAL PALSIES OF CHILDREN.

There are some points of difference between the cerebral palsies of children, and those of adults. In the case of a child we are dealing with a lesion of young and growing parts, and not of structures which have attained their normal form, and consistency. Causation is also different, since hemiplegia due to primary disease of the blood-vessels, congenital syphilitic endarteritis of course being excluded, is as rare in childhood as it is common in adult life. Most infantile and birth cases are due to prenatal changes, or birth-injuries. A convenient form for graphically presenting the subject is Sach's table:

GROUPS.	LESIONS.
Paralysis of Intra-uterine onset.	Large cerebral defects. Porencephaly. Defective development of the pyramidal tracts. Cortical Agenesis, highest nerve-elements affected.
Birth-palsies.	Meningeal hemorrhage, rarely intra-cerebral. Later conditions are Chronic Meningo-encephalitis, Sclerosis, Cysts and partial Atrophies.
Acquired Palsies.	Hemorrhage, meningeal and rarely cerebral. Thrombosis from endarteritis and Marantic conditions. Embolism. Resulting conditions are: Atrophy. Cysts, and diffuse and lobar Sclerosis. Chronic meningitis. Hydrocephalus rarely arises from this. Primary encephalitis (the Polio-encephalitis corticalis acuta of Struempel?).

It may be stated in this wise: If the mother is diseased, she may set up a process in the child which will prevent the development of the brain. From this will ensue a paralysis, discoverable at birth, or perhaps not until the descending tracts are called upon for function, when either they, or their nutrient cells, or the whole axon, will show an inability to fulfill the demands of life. The child may be injured during birth, and will be paralyzed at once, or in the immediate future. Later in life, within the first six years, as the result of an accident, or secondary to some of the infections, paralysis may result. In reference to this later division, it must be kept in mind that paralysis which results from the infections

may occur later in life, but it will not give rise to the infantile form, since by this time the brain and cord have reached their full developement.

SEX. This is equal in the infantile forms, but there are more males in the birth palsies.

For purposes of description it will be advantageous to treat under one division the Congenital and Birth-palsies, and make a separate one for what are distinctively known as the Infantile Palsies.

CONGENITAL AND BIRTH-PALSIES.

ETIOLOGY. These must again be considered under separate heads, since etiologically they differ. Congenital palsies have occurred from injuries to the mother; they usually arise from diseases of which the mother is a victim; maternal syphilis has been urged as a direct cause, but it more generally acts in the manner of hereditary syphilis; palsy has often been noticed in children born before term, and in such cases the paralysis points to a lack of embryonic developement. They are practically unfinished children.

Birth-palsies present a different origin. In these cases we are struck by the fact that male children of elderly primiparae furnish the majority of the cases, and it is evident that a disproportionment between the head and the birth-canal is a definite cause. An abnormally narrow or deformed pelvis is apt to produce it, and the mere prolongation of labor has been followed by it. In this latter case it is attributed to direct intoxication of the cortical cells by the retained Carbon dioxide. Too rapid a delivery has at times been followed by paralysis. In this case it is probably due to the skull being softer than normal. The most potent cause in modern opinion is the too vigorous, or the unskillful application of forceps. While we are concerned mostly with cerebral palsies, it must not be forgotten that spinal palsies are, more rarely it is true, produced in the same way.

PATHOLOGY. This must also be treated under the separate divisions. In Congenital palsies hemorrhages and areas of softening are sometimes found, but usually the paralysis follows less massive accidents. The usual condition discovered has been a diseased condition of the cerebral vessels, which has deprived the adjacent part of the brain of its nutrition in the period when rapid developement should have been the rule. The brain does not develop until the 9th intra-uterine month, the motor tracts until 1st to 2d month after birth. As a result we find patches of sclerosis, pointing to a condition of possible Meningo-encephalitis, or a loss of tissue in the greater part of a convolution, or even part of a lobe, producing the condition known as Microgyria, or causing

the formation of cavities, sometimes of large size, and communicating with the ventricles, which condition is termed Porencephaly.

Birth-palsies result from hemorrhages from the veins as they are about to empty into the great longitudinal sinus, but they may come from numerous small vessels distributed over large areas, or, more frequently, from small pial vessels on the surface, and from vessels within the substance of the brain, and the cord. It will be basilar in vertex presentations, and vertical if the breech presents. If it is over the motor area, we shall find a paralysis; and if over the frontal lobes, we shall have idiocy as a result. From the fact that it usually occurs at the vertex, we are most certain to get a paraplegia, and as the blood by gravity and compression flows down over the brain surface to a varying distance, we are liable to have a diplegia. Under such conditions, it is almost certain that it will flow lower on one side than on the other, and therefore it is common to find one arm, or arm and hand, more affected than the other, and to have varying degrees of speech-defect. If the amount of hemorrhage is large, there will be immediate destruction of brain or cord tissue, as in an apoplexy of adults, while if it is small, or punctuate, we shall get sclerotic patches or areas. In the cord we may get Syringomyelia.

SYMPTOMATOLOGY. The characteristic loss in the Congenital and Birth-palsies is Diplegia, and Paraplegia, while Hemiplegia is rare. When it is congenital and paraplegic it is the typical Little's Disease, which is an evidence of lack of intra-uterine development, to an extent that we must term it an unfinished child. They commonly weigh less than four pounds, and the birth is generally premature. Hence there is good ground for considering the symptoms at birth as results of imperfect fitness for life, rather than of injury at the time. In these cases there may be almost every combination of paralysis, except a distinct paralysis of the face. At birth the congenital case may be found to be incapable of motion in the lower limbs, and upper as well, perhaps, but there may be no obtrusive sign of any paralysis for weeks or months. The common phenomenon in these cases is a prolonged period of asphyxiation, and a difficulty in swallowing, while convulsions are quite rare (contrary to the rule in birth-palsies).

The cases resulting from birth injuries have a different history. While the paralysis is not present for some time, instead of being found at birth, if careful investigation is instituted, a difference in the motor power of the limbs will be discovered; convulsions begin at once, or within a few hours, or a day or two after birth, and may continue for several days and then never recur, or they may recur at intervals for years, or may continue through life. These come from irritation of cells which have escaped actual destruction. The motor loss, when found, is the same in possible distribution, but is flaccid in the congenital form, whereas there is rigidity in

the birth-palsy. In the birth-palsy, when the child attempts movements with the legs, they will be found to be weak and awkward, and almost universally the first obtrusive fault will be an adductor spasm which holds the legs together. The pathology of this spasm, which is a prominent symptom in all these palsies, is extremely doubtful, since its occurrence does not seem to depend very closely upon site or area of lesions. Gowers thinks that it depends less upon the injured parts, than upon the necessary control, which now is lacking, of the parts unaffected. It certainly follows lesions of the central ganglia, as well as of the cortex. There is the same spasm in the calf-muscles, and all conspire to make the leg rigid, and the stiffness is increased by attempts to bend it, so that the leg is moved as a whole. Another early condition in the birth-palsy is rigidity of the arms with an inversion of the thumbs, and the head may be bent back. If the rigidity is one-sided, the convulsions are also. In both varieties of palsy, walking is long delayed, if it is ever possible, on account of the adduction, and even crossing of the legs (scissors-legs). When the child does start to walk, we find that it teeters on account of the foregoing disabilities, and the contractures which result, and also because movement of spastic muscles increases spasticity. This gait is called titubation. These contractures produce in the legs talipes of some sort, usually equino-varus, but the inversion may be so great that the soles of the feet point toward one another. Spasm results in hypertrophy of the muscle involved, and so some muscles will be very weak while they are enlarged, but the muscular masses as a rule are diminished in the affected parts. When this spasm is very marked, an attempt at extension of the leg will set up an irritation of the muscles, and after a little extension the limb will jerk back. The spine may become curved by the irregularity of the muscle-supply and spasm. The knee-jerk is exaggerated, and ankle-clonus might be expected, but will be absent in those unable to walk, since the condition of extreme muscular irritability neutralizes the reflex.

Athetosis, and a finer movement which has been termed hemiplegic chorea, and tremor are also found. Athetosis consists of queer worm-like movements of parts which may begin in one part, and then go to another, and then another, or may be confined to a single part. This may be all over the paralyzed parts, and even in the face, and the platysma, but is more often in the fingers of one or both hands. These cases show an over-extension, and subluxation of the first phalanges from spasm of the interossei, and that allows the fingers a wide range of movement, which may resemble our idea of gyrations of the snakes on the head of Medusa, more than any movement of those members, with which we are familiar. This condition sometimes renders the affected person entirely helpless, as the hands and arms are unsuited to any form

of labor. There is a finer range of movement which is called chorea, although that name is also applied to athetosis. Some subjects display a tremor which begins with a movement, and grows constantly coarser and wider as the movement is developed, until it renders the motion as abortive as the intention tremor of multiple sclerosis. This has been called chorea spastica, and both it and athetosis are common in the hemiplegic form of paralysis. There is no loss of sensation of the skin.

Gowers mentions seeing one case where there was a considerable defect of sight without any change in the ophthalmoscopic image. Squint is not uncommon, and seems to arise from spasm or weakness of a variable location, first affecting one, and then another muscle.

The sphincters of the bladder and rectum are not paralyzed, but may be weakened, or may be the site of spasm. The mental state being low, initially at any rate, combines with their muscular losses to make them filthy in early life, but they tend to improve as they grow older.

Swallowing is initially faulty in the Congenital cases to some extent, but it is not a feature of later periods, although they drool saliva from inability at first, and from habit perhaps in later years. There is over-action of the salivary glands in some cases and the habit, which is well-nigh universal, of continually thrusting their hands into their mouths, sometimes for hours together, all contribute to the same result.

Speech is not normal in any but the very mildest cases, and the loss ranges all the way from a slight defect, which is almost completely obliterated as time goes on, to a creature who can only signify his wants by gibberish totally unintelligible to any but his constant attendants. The amount of aphasia is closely related to the extent of the mental loss.

Mental loss can be said to regularly follow the extent of the paralysis. A diplegic is prone to idiocy, while a paraplegic may be precociously bright, but it is safe to say that they are never quite normal mentally. Hemiplegics differ among themselves, showing all grades of mental equipment. It may be broadly stated that severe physical loss presages great mental deficiency, but the converse is not equally true.

Epilepsy results from secondary degenerations; it is not so common in these as in the infantile cases, but its advent indicates that at length the mind will fail clear to dementia or insanity.

Electrical reactions are normal, and the atrophies are only slight, and on the contrary pseudo-hypertrophy may ensue in the affected muscles.

INFANTILE CEREBRAL PALSY.

DEFINITION. This is a paralysis of cerebral origin occurring in the first six years of life. Gowers says that it is equally frequent in the first two years, three-fifths of all cases occurring then, while three-fourths occur in the first three years, and seven-eighths in the first five years. It is always a Hemiplegia, and the cases which affect both sides of the body will be found to be no exception to this rule, but are simultaneous lesions upon the hemispheres. This variety of paralysis is separated from those occurring after the age of seven by no fanciful distinction, but upon the real basis that this is a definite stage in the process of development of a child. Cerebral injury at this time produces a halt in the development of an affected limb, which is not true of a similar accident at a later period, and since, up to the age of seven, the speech center is not so firmly localized on a single side of the cerebrum, permanent aphasia is almost unknown if the lesion is single, whether it be upon the left or the right side of the brain.

DIFFERENTIAL DIAGNOSIS. The differential diagnosis of the Infantile form is from Paralysis from Caries, Anterior Poliomyelitis Acuta, Cerebral Palsy from Tumor, Spinal Sclerosis (congenital), Myopathy.

SEX. Female children are more prone to this condition than males.

ETIOLOGY. Few die from this disease, and therefore the pathology is inferential to some degree, and on that account some, Taylor of London for one, have separated the cases into the following classes: those paralyzed secondarily to some infectious disease, and those paralyzed from a cause assumed to be primary. Since the symptomatology is not assumed to differ, it seems to sufficiently satisfy the conditions if we say that such paralyzes are apt to occur during convalescence, or a short time after it, in a patient who has suffered from some one of the infectious diseases, of which scarlet fever and measles are the most potent. Cases have been noted after diphtheria, pertussis, bronchitis, gastric fever and mumps. It has come on a day or two after a child has suffered from a fall. A majority of the cases (5 out of 8, Gowers) have been preceded by no apparent infection, and constitute the so-called Primary cases which differ in no manner from the others, either in method of onset, progress, or subsequent results, and therefore rest upon a similar infective basis, although its point and manner of entrance is not susceptible of demonstration.

PATHOLOGY. Our actual knowledge is slight, since, as has been said, few die in the early stages. The lesions found have been a superficial sclerosis of the hemispheres, microgyria, and porencephaly. The usual effect of an infective agent is to produce thromboses and emboli, and that is the theory held by Gowers as

to the process in both the primary and secondary forms, and of these he favors thrombosis as being the most probable. The occurrence of microgyria or porencephaly would rest then upon the occurrence of a vascular obstruction in a vessel of the surface, or in the mass of the hemisphere. Struempel considered that the cause of the primary cases was an acute polioencephalitis. Both Gowers and Sachs point out that the scanty post-mortem findings have not disclosed any such condition; that pathology teaches us that a primary inflammation of the cerebral nervous tissue is far less common than of the spinal, and that analogy from spinal cord diseases is not a good guide, since the spinal nervous system is not susceptible to secondary disease, while the cerebral is eminently so.

SYMPTOMATOLOGY. **Primary Effects.** The illness begins with or without fever, but in 50% there is a severe convulsion, or a series of them, which may be one-sided at first, and afterward general. They may persist for days and hours, or may cease for a time and again recur. Soon after this the child is found to be paralyzed upon one side; if on both it is a double lesion. This paralysis will be found to be on the side on which the convulsions manifested themselves, or where they began and were most severe. Vomiting may be present. This paralysis is a permanent one, but its area may be quickly reduced. After a few months it has become very much reduced, and the face and arm are apt to be comparatively free, on account of the power of compensation from the hemisphere opposite to the site of attack. In quite a proportion of the cases, however, some traces remain in the arm, but the shoulder and elbow are less liable to be disabled than is the hand. If the case is secondary to some infectious disease with much prostration, the patient may be so weakened by the shock that the paralysis is not discovered for some days. Unilateral lesions have not quite the localizing value that they have in adults, since the lesion may lie in the central ganglia. The leg always regains some power, although under the most favorable conditions there will be some limp, on account of the arrest of a normal amount of developement. The face-palsy is generally trifling, and the only trace may be an overaction of the paralyzed side. This is best disclosed by some response to the emotions, where the movement will begin first on that side, while a strong volitional movement will not disclose any difference. Paralysis of a lasting nature never results, unless the motor cortex or tracts are directly injured.

If speech has been developed prior to the seizure its loss will only be temporary, and will be regained in a week or two, or at most in a couple of months. Sensation is not affected.

Knee-jerk is exaggerated, and ankle-clonus is frequent (contrary to the rule in birth-palsies), but rigidity is not so much a feature as in adult cases.

Mental changes may be as great as in birth-palsy, and they may be present, although the degree of paralysis may be slight. We may find every variety of mental obliquity, from hysteria to idiocy, and Gowers states that the worst case of hysteria that he ever saw was in a girl who had suffered from infantile hemiplegia.

LATE EFFECTS. When the convulsions with which the attack was ushered in are accompanied by loss of consciousness, with tonic and clonic spasms, and occasionally with biting of the tongue, and relaxation of the sphincters (they may resemble petit mal), the resulting paralysis is usually slight. In such cases a usual result is Automatism, which is the performance of acts without apparent volition, and often is important in a medico-legal way. Convulsions are recurrent at a later period in life in two-thirds of the cases, and resemble idiopathic epilepsy, although the etiology is different. These subsequent convulsions may consist only of a loss of consciousness, or they may be typical epilepsy even to the aura. These attacks start like a focal epilepsy. The convulsions may begin years after the attack, or be seemingly excited by puberty, to recur at intervals permanently thereafter; almost always one-sided, and affect only the paralyzed limb or that whole side.

Contractures come on as power returns in a limb which has been paralyzed, and spasm is found in various muscles. There is a continued, but perhaps slight spasm in the feet, which can always be overcome by gentle pressure, but which is sufficient to over-extend the toes, and cause the person to be uncertain in gait (titubation), even if he have the requisite muscular power. It produces some grade of talipes varus, or equinovarus. The same condition is seen in the middle joints of the fingers, producing over-extension, and even a subluxation of the fingers. There is a fixed spasm at times in the elbow, and the wrist in extension.

Athetosis is seen in from 10 to 50%, and if an adult is found with this symptom it is almost certain that the paralysis from which he is suffering is of infantile origin. Not exactly the same, but similar, are what are known as associated movements. These are such movements as are repeated on the well side, when a strong effort is made on the paralyzed one.

It may be generally stated that voluntary movements are disordered and ataxic.

Atrophies, accurately speaking, are not a symptom of this disease, but there is an arrest of developement which gives that appearance to a limb. The presence of an undeveloped scapula and arm are diagnostic of infantile paralysis. The muscles are usually reduced in size in the affected area, but an exception is found in those which are the seat of spasm, since its continuance usually produces hypertrophy of the affected muscle.

PROGNOSIS. The danger to life is not very great, if we except

the cases which succumb to the severity of the initial convulsions, or, less often, to the strain of the status epilepticus in later years. Mills differs in saying that most diplegics die in infancy or childhood, while Oppenheim says that they live to an old age. All tend to show some physical improvement, but it differs in the different types, although some cases of even the worst type will almost completely recover. The greater the defect, the later does improvement take place.

Diplegics have the worst prognosis, since the symptoms which make us take a pessimistic view of any case are most frequent, and most marked, in this variety. These symptoms are convulsions, contractures, mental enfeeblement, athetosis and epilepsy. The history of the first year gives us grounds for predicting the future, and yet some of these cases begin to develop mentally after puberty. It is not possible to give a definite prognosis about the power of walking until after the sixth or seventh year, since some children will finally walk fairly well even if at first the progression is cross-legged. If the paralysis comes on after birth, i.e., is typical infantile palsy, the probability of mental impairment is greater, the younger the age at the time of the attack.

Aphasia has a good prognosis where it is not accompanied by mental defect, since in early years the speech-center is not so definitely and exclusively localized in the left hemisphere. Dysarthria may remain. This may be helped by training.

Epilepsy is not liable to occur, if it does not occur in the first two or three years, but, exceptionally, it has been known to delay its advent until the tenth year. Its appearance presages eventual idiocy, and at any rate great mental impairment. Its attacks seem to grow less in mature years, and it is said to disappear about the fifth decade.

Athetosis and choreiform movements are the cause of great disability, inasmuch as they may render the hands and arms utterly useless. The appearance of these and the contractures render the outlook very gloomy, since they do not tend to pass away when once established. Bilateral chorea has an especially bad prognosis. Mere retardation of mental powers and speech should not in itself be a ground for a bad prognosis, since recovery of these may come later, but mental impairment, if continued, carries with it a probability of physical defects.

Convulsions in a birth-palsy are a ground for prognosis, depending upon their severity and persistence, since they show the depth and area of the brain injury.

In general we may say that the prognosis is good, if, after a few months, spasticity is still absent in the limbs, and there are some attempts at voluntary movement.

Paraplegic cases have a better outlook, since speech-defect is usually only associated with implication of the arms, and these

cases also do not have much liability to mental losses. While the legs may be stiff and uncontrollable, patient treatment will often improve them greatly, even if walking is delayed until the sixth or seventh year. These cases are subjects for hopes of betterment, however, rather than of perfect recovery.

In all cases, the presence of the stigmata of degeneracy causes one to make a less favorable prognosis, and a microcephalic head debars us from giving much promise of further development.

DIAGNOSIS. The presence of a paralysis, either diplegic, paraplegic or hemiplegic in localization, and with exaggerated deep reflexes, proves that there is some injury to the motor tracts above the motor cells of the anterior horns of the cord. This makes it an upper neuron paralysis. The implication of speech, or the mental faculties, assures us, among other symptoms pointing to the same localization, that the cells of the motor cortex of the brain are primarily affected. We are sure then that the case is one of cerebral paralysis. If the history was faulty or absent, the deficient development of the affected parts, the presence of athetosis and associated movements, deficient mental development and recurring convulsions, or some form of epilepsy, would be strongly indicative of an infantile origin. On mere localization of the paralysis we should have grounds for assuming that a diplegic indicated a congenital case, that paraplegia was a birth-palsy, and that a hemiplegia was infantile. Idiocy, as well as cranial asymmetry, is indicative of prenatal origin, while epilepsy and recurring convulsions are more common in the infantile cases, and a paralysis confined to the lower limbs (paraplegia) with an unimpaired intellect is often found in cases of birth-palsy. If we have a history, we should conclude that a paralysis was prenatal in origin, if, after a normal birth, the child was deeply cyanosed, or, without previous convulsions, or feverish attack, should display later an extensive paralysis, particularly if it was a diplegia; that it was a birth-palsy, if an infant, after a difficult, precipitate, or instrumental birth was at once, or very soon, seized with convulsions which were long-lasting or recurrent, after which a paralysis was discovered; that it was infantile, i.e., before the seventh year, if, after a history of good health, the child became the subject of a hemiplegia, without warning, or during convalescence from an infectious disease, even if some time had elapsed from apparent recovery, and if the aphasia accompanying it was very transient.

DIFFERENTIAL DIAGNOSIS. Spastic Paralysis from Caries of the Vertebrae may closely resemble cerebral spastic paralysis, but in this case the sufferer from caries had a history of normal physical power, and an examination of the spine will reveal a tender area where motion is restricted, and there will usually be disturbance

of temperature, and localized pain. A skiagram will reveal pathological changes in the spine.

Anterior Poliomyelitis. The patient here has also been normal. There is generally, although not always, a history of a febrile attack preceding the palsy. There is also marked wasting of distinct muscles, while others remain plump and well-nourished, and it is a flaccid, and not a spastic paralysis; there is loss of deep reflexes, and of the reaction of the muscles to faradism. A whole limb is not so evenly affected.

Spastic Paralysis from Tumor. When the history reveals that the paralysis has been gradual in onset, and without symptoms of an acute antecedent disturbance, the presence of a tumor should be regarded as a possibility.

Spinal Sclerosis, (Congenital.) In some cases the damage to the nervous system of an infant in utero is expended upon the spinal, instead of the cerebral structures. Generally this takes the form of congenital syringomyelia, and is not likely to be confused with the diseases under discussion, but two cases are noted by Dejerine which show that such a localization may be a source of error. The first case was diagnosed as a congenital spastic quadriplegia, lived until the age of 63, was intelligent, and was supposed to be suffering from the effects of a double hemispherical lesion. On autopsy was found to have a spinal syphilitic lesion only, the brain being normal. His second case also proved to have a normal encephalon. The diagnosis rests, in a spinal localization, on integrity of the intelligence, absence of paresis and spasm of the face, and absence of convulsions.

These may also be the conditions in a typical Little's Disease, if the arms are not involved. If they are, we shall have involvement of the face, strabismus frequent, intelligence affected, and epilepsy frequent.

Myopathy may look like it, but it is not hemiplegic, the knee-jerks are not exaggerated, there is no history of an acute attack, nor have there been any convulsions.

TREATMENT. This is divided into several groups on account of the difference in method of onset, and also from the fact that there is no relation between the measures necessary to antagonize the early, and the late features of the condition. When the palsy is the result of some pre-natal condition which has run its acute course before the birth of the child, our treatment will be that of antagonizing results. If it is a case of birth-palsy, the infant will suffer from convulsions at its birth. These are recurrent, and very obdurate, the mortality is extremely high, and there is little scope for symptomatological prescriptions. If a few doses of Belladonna, Arsenicum, Cuprum ars., or Hyoseyamus as indicated do not relieve them, more drastic measures should be employed. Compression of the carotids against the hyoid bone will reduce the

flow of the blood to the brain; the pressure should be continuous, but not persisted in for more than an hour. Chloroform and Amyl nitrite have been used, but not with very favorable results. Cold applications to the skull are not of much use. Chloral hydrate, 2 grains, and Potassium bromide, 5 grains, were given every half-hour to two hours to a case of my own, with good results. The hot-bath is not indicated in this condition, but in cases of doubtful etiology it can do no injury. When the case is infantile, and at a later period of life, the treatment should be that of an apoplexy in an adult. In these, as well as birth-cases, great care should be exercised that position should never be such as to impede the venous return from the brain. If there are good grounds for the localization of the clot, the skull should be opened, and the clot washed out. It is advisable to take long chances in this procedure, since the hemorrhage is meningeal, and the after history of these patients, if they recover from the initial shock without operation, is one of harassing invalidism, since they surely will have some grade of spastic paralysis, and generally will also be epileptic. The treatment of the late stages of these cases is the same for all, as it comprises all the possibilities when we conserve the general nutrition of the body, the special nutrition of the limbs, combat deformities, and mitigate as much as possible athetosis, spasms, and epilepsy. The general care should involve the most assiduous training of mind and body, and sometimes results more happily than we should expect, from the ingenious substitution of normal parts in the offices of the impaired ones. In this department, as well as in the more specialized ones, it will often be found that the constant care of a mother will accomplish more than the occasional treatments of a more skilled operator. The nutrition of the limbs may be very greatly improved by massage several times a day, immersing the limbs in hot water for ten minutes before the massage, and by faradism to the extensors in the upper limbs, and to all the muscles of the lower. The feet should receive great attention, since abnormal positions of the toes and feet are often a great hindrance to walking. Phosphorus 6x, is a remedy which may be of use in this connection. Much can be done by these means, if education is co-incident, and painstaking. All this must be begun at an early date to be of much value. Contractures should be antagonized by splints, and tenotomies, and lengthening of the tendons, and this must be early in the birth-cases, since they do not respond as well as do the infantile. Tendon-transplantation has not been of as much value as in the contractures of the adult, but the substitution of normal muscles for impaired ones by tendon transplantation results more happily than one would expect. In the use of electricity the applications should be made with no current until the child is free from terror at the procedure, and then only such currents should be used as do not

induce painful shock. The cortical centers have been excised when they could be accurately located, and with the substitution of a flaccid for a spastic paralysis. Nerve-stretching, and plaster of Paris splints, are useful in the treatment of athetosis. Generally, however, it is irremediable. The efficiency of remedies is very doubtful, but some clinical reports lend color to the belief that they have mitigated conditions at times.

THERAPEUTICS. Baryta carb. This is a remedy for deficiencies in nervous tissues, and has as a symptom tension in the calves, and muscles of the back.

Causticum. Contraction of flexor tendons. Contractions of the tendons of the fingers and twitchings in them, and also in the tendo Achillis, with a marbled appearance of the skin. Contraction of the plantar muscles. It certainly does favorably affect the nutrition of nerve-cells.

Calcarea carb. The symptomatology indicates that this may be useful in the birth cases, and the ante-natal ones, where there is a faulty development of the motor tracts.

Plumbum. This has been recommended, but it has a flaccid, and not a spastic form of paralysis.

Secale. This remedy is indicated for a spastic form of paralysis, with a cold surface and hyperesthesia, so that touching the skin arouses twitchings and jerkings. There is a spasmodic curvature of the arms, with a distortion and turning back of the fingers, and the same in the legs. A convulsive spreading-apart of the fingers, with the other symptoms, might point to its use in athetosis.

Silicia. It is a specific for the re-absorption of scar-tissue in nervous structures.

Sulphur. This is on constitutional indications, and is not to be ignored.

Lathyrus. This produces a general spastic condition.

Graphites may prove efficient as an absorbent.

The late epilepsies should be treated as is indicated under that caption.

TUMOR OF THE BRAIN.

DEFINITION. This is the formation of new growth in the brain, and the term is elastically enlarged to include growths in the cranial cavity which compress or irritate the brain, without actually being developed on, or within it. All the common forms of tumor are possibly located here, and some are peculiar to this locality.

SYMPTOMS OF A TYPICAL CASE are headache, double choked disc, vomiting and vertigo, with retardation of the pulse, generalized spasms, somnolence and mental dullness.

DIFFERENTIAL DIAGNOSIS from Chronic Diffuse Nephritis or Contracted Kidney, Lead Intoxication, Hypermetropia in Cachectic Girls, Profound Anemia, Amenorrhea, Hydrocephalus, Acute and Chronic Encephalitis, Acute and Chronic Generalized Meningitis, Alcoholism, Cerebral Palsies of Children, Hysteria, Brain Abscess, Cerebral Hemorrhage, Intra-cranial Aneurism, General Paresis, Disseminated Sclerosis, Multiple Neuritis (alcoholic and cachectic), Epilepsy, Migraine and Melancholia.

AGE. It affects persons of all ages, from the sixth month to the sixtieth year, but definite differences characterize the growths which will probably be present at the different periods of life. Tubercle is most probable in childhood, and decreases in frequency as the years go by, until it becomes highly improbable after the thirtieth year, although it has been found at the sixtieth. Sarcoma, glioma and syphiloma occur at any age, but are the tumors usually of early middle life.

SEX. Males are more often the victims than females, and this may depend upon the etiology.

ETIOLOGY. This is often a matter of conjecture. Cicatrices can often be found at the site of origin of a tumor, but they must be supposed to have a previous injury as their determining cause. Injury, therefore, even if remote, is considered to be a common cause. They sometimes arise from anomalies of development, and gliomata most frequently are the result.

PATHOLOGY. Gliomata are not as common as has been supposed, since they are actually the third in point of frequency. They are less frequent than either tubercle or sarcoma. They are almost always single, and are always primary, except when an extension from glioma of the retina. They vary in size from that of a hazelnut to that of one's fist, and are yellowish-white to a reddish-gray in color. They often closely resemble the normal brain-substance. They are softer than a sarcoma, are very vascular, and if hemorrhages have occurred, and such hemorrhages are of frequent occurrence, they may be taken for spots of softening. They may resemble a local hypertrophy from the similarity of their color and consistency to the normal brain. The hemorrhagic tendency makes them liable to contain cysts, and it is held by some that all cysts in the brain, which are not the results of hemorrhage, or softening, are features of a diffuse glioma. Cavities lined by cells are, however, rarely gliomatous. They extend by infiltration, and not by compression, and their rate of growth is slow, often coming to a standstill, and if rapid is almost always the result of a hemorrhage. Their special localization is in the hemispheres, or the cerebellum, more in the white than in the gray matter, and they are rare elsewhere. They do not involve the meninges or the bones.

Sarcoma. This is next to tubercle in frequency, always single,

except when in the shape of melanotic nodules, which form is practically unknown in the brain. They may, however, be diffused over a considerable area, but resemble in size the gliomata, while harder in consistency. They may be metastatic. Sometimes they undergo caseation and fatty degeneration, and cannot always be distinguished in gross appearance from glioma. They are non-vascular, and so hemorrhages and apoplectic attacks are rare in their symptomatology. They act by compression instead of infiltration, are always surrounded by a capsule, or by a zone of softening from the brain substance. Infiltration is a possibility only. They may commence in the brain-substance, but almost universally develop from the periphery into the brain-substance. The sites of election are the cortex and the cerebellum, but the actual point of origin is the meninges, the periosteum and the bones, and there is often a diffuse sarcomatous condition of the base of the skull, temporal and sphenoidal bones, and less often of the occipital. The meninges are also a frequent site of diffuse sarcomatous change. They are the most operable of all tumors.

Tubercle is the most frequent of all tumors, and is multiple as a rule, and practically always metastatic. Solitary tubercle is also found (20%), and is similar to a gumma. The tumor is often accompanied by a miliary tuberculosis of the neighboring meninges. This is diagnostic if present. The tumors are small and rounded, and not larger than a goose egg. The growth is not vascular, and in the later periods is subject to caseation in spots throughout its mass. Its growth is rapid, but there may be periods of stasis, and even of regression. The peripheral portions have the characteristics of granulation tissue. No portion of the brain is immune from the invasion of solitary tubercle, but it is most common in the pons, cerebellum and the cerebral cortex. It does not infiltrate, but compresses. Its point of origin is the lymphatic sheaths of the blood-vessels. On account of its similarity to other neoplasms, the only positive diagnostic feature is the discovery in its tissues of the tubercle bacillus.

Syphiloma are rare in childhood, and are never the result of hereditary syphilis. They may appear at any period, from one to twenty years after the infection. The growth may be in the form of a tumor resembling solitary tubercle, but not reaching so great a size. Both these growths are non-vascular, and caseation is an incident of their later period. Multiplicity is the rule. The most usual form of syphiloma is that of a soft, gelatinous mass at the base of the brain, which mats all the tissues and structures together, and involves them in its contraction. When on the meninges it is of firmer consistency. No matter where it is evident, syphiloma almost always arises from the meninges, and a connection can be traced between it and the pia. The other point of origin is from the vascular walls. Sudden increase in symp-

toms, and as sudden a regression and possibility of cure, are the distinguishing clinical characteristics of syphiloma.

Carcinoma is usually found only in persons over sixty years of age, and is never found in children, unless as a metastasis from cancer of the orbit. It is a soft tumor of irregular outline, and is very vascular. Almost without exception, it is metastatic. It is one of the large tumors, and may reach the size of a child's head, and is ordinarily multiple. Its contents are colloidal, and it softens into a pulstaceous mass, and acts both by compression and infiltration. It may arise from the dura, or from any of the structures of the brain, and is rapid in growth. It may be circumscribed, or diffuse, and rather selects the central ganglia. It is the least operable of all.

Psammomata, cholesteomata, angiomas, and dermoid and parasitic cysts are seen at times in Europe, but are infrequent in this country. As they act only by compression, the symptoms are apt to be doubtful, and they may give rise to no symptoms. If they can be localized, they are removable, as they possess a capsule. They are often multiple.

LOCALIZATION. Tumors may arise anywhere in the cranium, but are most frequent in the medullary portion of the cerebrum, and next in the cerebellum. The pons is next in frequency, and then the central ganglia, and lastly the basal ganglia.

CHANGES RESULTING FROM THE PRESENCE OF TUMOR. In the description of the various kinds of tumors, it will be noticed that while glioma, tubercle and carcinoma infiltrate the brain-tissue, sarcoma, syphiloma and the unimportant varieties of tumor and cyst simply compress these structures. Some produce both effects, while others are too small to produce much pressure. Most symptoms are produced by compression. By this the convolutions are flattened, distorted and pushed from their places, and the pia and the brain generally are dry and anemic. The brain is softened, where a growth impinges upon it. A growth can scarcely be so small as not to interfere with circulation, and a wide range of symptoms is thus entailed. The cerebro-spinal fluid is increased, and hydrocephalus is a common accident, particularly in case the tumor is near, or in the cerebellum, since the flow from the great Galenic veins is impeded. Infiltration of the sheaths of the emergent nerves takes place, with resulting changes in them, and they are also flattened by the pressure. This is most marked in the abducens and the olfactory. The spinal cord may be the site of changes, both from pressure and interference with circulation. Pressure, both local and general, may thin out, or even perforate the skull, and the sutures are usually separated in young children.

SYMPTOMATOLOGY. This is general and focal. The general symptoms, which inform us that there is a tumor in the cranium,

are headache, double choked disc, changes in disposition and mental power, stupor, vomiting, vertigo, retardation, or other changes in the pulse, changes in respiration, generalized spasms, insomnia, polyuria and emaciation. General symptoms are early, and are the cause of most of the distress.

FOCAL SYMPTOMS are unilateral spasm, monoplegia or hemiplegia, paresthesia or anesthesia in one or more limbs, hemianopsia, the various forms of aphasia, affections of the cranial nerves, and the basal portions of the brain.

GENERAL SYMPTOMS. Headache is an almost certain and constant symptom, and is generally the initial one. It is not always initial, or constant, but recurrent, and few patients escape it. It is usually diffuse, and not of particular localizing value, but is more apt to be severe if the growth is under the tentorium. If combined with superficial tenderness from percussion, and especially if accompanied by a bruit, it may localize a tumor of the cortex. It is produced by a stretching of the sensitive dura, and the branches of the fifth nerve, and is most constant and severe, especially at night, in syphilitic growths. If very severe, and extending down the back, it suggests a growth in the posterior fossa, or in the cerebellum. Children suffer less from it, on account of the ease with which separation of the sutures is produced. It is aggravated by anything which suddenly increases the pressure within the skull, like coughing, sneezing, or sudden changes of position.

Choked Disc. (See cut on page 103.) This is the condition where the head of the optic nerve becomes congested, and opaque, and loses its clear outline. Its typical termination is in optic atrophy, and loss of sight, but this is not always accomplished, and loss of sight may be such a late result that the ophthalmoscope is a necessity for its recognition. In children we are more apt to find loss of vision as a primary symptom. Choked disc may antedate the headache. Primary optic atrophy is never found in tumor. It is always secondary, and indicates a tumor of long standing. Choked disc occurs in from 80 to 90% of the cases, and is almost always bilateral, although it is often more marked in one eye than in the other. It may be present in a high degree without impairment of vision, and is always present in tumors of the corpora quadrigemina, in 89% of tumors of the cerebellum, and absent in two-thirds of the tumors of the pons, medulla, and corpora callosa. It is least frequent in tuberculous, and most common in glioma and cystic tumors. In the rare cases where it is unilateral, it is ascribed to the location of the tumor in one nerve, or in front of the chiasm. It is produced, probably, by an inflammation resulting from the forcing of the cerebro-spinal fluid into the sheaths of the nerves. This sets up an interstitial inflammation which later affects the nerve fibres,

but initially stops the venous return. It may result from the action of toxic products released by a primary inflammation of the disc.

Mental Changes. The mind usually shows the effects of cerebral pressure in a change of disposition, generally a mental dulling, but sometimes in irritability. The patient may be melancholy, and seek solitude, or he may be delirious and excited. The cause may be compression, but in the absence of any general symptoms, the patient may display a peculiar mental weakness with exaltation. It might be ascribed to the causes producing headache, but has been seen in its absence. Some change is almost always present. I saw a case, however, in which, until a late period, there was undisturbed mental ability, and unimpaired equilibrium.

Stupor. A stuporous condition is very characteristic. It may be present at all stages, and the patient will first appear sleepy and apathetic. He will next actually fall asleep, if not especially interested; later he will fall asleep while talking or eating (Narco-lepsy) and in the last stages there is a condition of lethargy approaching coma. The patient can be aroused with difficulty, and is apt to defecate and urinate unconsciously. This symptom may be absent if the tumor is very small, or if it lies outside the brain.

Insomnia. Insomnia is liable to be a symptom at some stage of a case, since it is the result of irritative and inflammatory conditions, which are extremely liable to be part of the pathological changes. It is the opposite of somnolence, which is a compression symptom. The insomnia may also be the result of the intensity of the general symptoms. It is more often found in adult cases than in those of children, and most frequent and pronounced in those arising from syphilis.

Vomiting. This is not always seen, but is rather dependent upon the rapidity of the growth of the tumor, or upon the fact that it is localized in the cerebellum or pons. It may be a symptom of tumor in any part of the brain. It does not become prominent until the headache has become a factor, and is often an accompaniment of its acute exacerbations. It is most common in children. It is cerebral in type, in that it is often unaccompanied by nausea, does not depend upon the character or quantity of food, is very sudden and violent, and accompanied by great prostration, and is projectile. Change of position may bring it on, and it may be so continuous as to threaten life by interference with nutrition.

Vertigo. This is a symptom of irregular appearance, and is quite dependent upon the location of the growth. It is peculiarly marked in cerebellar tumors, and often with those of the pons, and structures in the posterior fossa, because they compromise the functions, directly or indirectly, of the auditory nerve. While

actual vertigo, where the patient reels and must grasp objects for support, is uncommon, he is very commonly subject to an unsteadiness like the results of moderate intoxication. Equilibration is so complex a process that localization of a disturbing cause is very difficult. These patients may escape vertigo, or it may be an intermittent symptom, but they all complain of a feeling of pressure, pulsation, and discomfort in the head. They may have a vertiginous condition from implication of the motor oculi, auditory from implication of the 8th, and forced movements and attitudes from tumors in the middle lobe of the cerebellum.

Irritation of the dura is almost a necessity of the existence of a brain tumor. The nerve supply of the dura is mainly from the fifth nerve. A powerful peripheral stimulation of a nerve irritates its nucleus, and on account of the inter-relation of all of the nuclei is liable to injuriously affect the more sensitive ones. In this case it is probable that a secondary irritation of the vestibular and pneumogastric nerves is the cause of the vertigo.

Circulatory and Respiratory Disturbances. Fever may occur from inflammatory changes in the brain or meninges, and it has been said that local elevations of temperature can be discovered on the scalp over the growth. This is questionable, but possible if the growth is cortical.

The pulse is slowed (compression symptom), as low as from fifty down to thirty beats per minute. It is apt to be irregular. A fatal termination, or a direct action upon the vagus, is indicated by rapidity of pulse rate after a preliminary retardation.

Respiration is usually affected in some manner. It is apt to be slower than is normal, and subject to periods of irregularity. It may be Cheyne-Stokes in character at times, and then resume a more normal rhythm, but that condition usually presages a fatal termination.

Focal Symptoms. These are the symptoms from which we seek to localize the lesion which the general symptoms have led us to think to be tumor. These are of two varieties, direct, and indirect. The anatomy of the brain must be fully grasped in order that we may appreciate that one symptom can be due to the direct pressure of the growth, while another is manifested by the disturbance of a distant center, from the fact that the growth, which caused the first symptom directly, has also caused the second by pressure upon the motor tract leading to the distant center referred to, and to which the motor or sensory disturbance must be referred. It is then a question which is the direct, and which the indirect symptom. This is decided by the order of appearance of the symptoms, and also from the fact that no direct focal injury is without associated symptoms of a disturbance of contiguous areas. Slight pressure symptoms indicate a small tumor and that the focal symptoms are direct. Tumors are first to be

localized in the motor cortex, or at the base, before a finer localization is attempted. If at the base, there will be some degree of implication of the cranial nerves.

Focal symptoms are few, and they will always be the same for a tumor in identical localities, and they will be unilateral if the tumor is single; but multiple tumor is not unknown. They are unilateral, since no tumor is near enough the middle line to produce bilateral symptoms, and tumors even of the corpus callosum rarely produce bilateral effects. Tumors may attain large size, and produce no focal symptoms, if they grow slowly, and act by compression rather than by infiltration.

Frontal Cortex. Such a localization is probable if we have pronounced general symptoms, and an absence of focal ones. Changes in mentality are not focal symptoms, since this function is probably diffused over the whole brain, but it is considered that *early* mental loss, the appearance of a mild dementia without delusions, change of disposition, personal uncleanliness, and a loss of the sense of decency should influence the diagnostician in localizing the growth in the left frontal, since such losses appear *late* in tumors of other localities. This has been marked in tumors of the frontal cortex, and underlying white matter, but is not found in tumors at the base of the anterior fossa. The only definite symptom is an aphasia of gradual onset, and slightly pronounced character, and a reluctance to speak. This localizes the growth in the cortex of the left third frontal in right-handed persons, and in the right in left-handed. A tumor in the frontal cortex may produce motor spasms and paresthesias by a propagation of impulses to the rest of the brain, and there may be a disturbance of equilibrium, since here are located cells whose fibres go to the cerebellum of the opposite side. If the tumor is in the lower surface of the lobe, it will compress the optic and olfactory nerves, and will interfere with the sense of smell, and may produce hemianopsia, and a severe form of optic neuritis. If it invades the orbit, the globe will protrude and be paralyzed. Conjugate deviation of the eyes with muscular spasm in the arms or face make very positive a diagnosis of a tumor of the frontal lobe. The eye symptoms with rigidity of the neck and back imply the same condition.

Motor Area. A tumor of this area may be cortical and give rise to very definite symptoms, since the functions of every part of this area are so well understood, or it may be so far down in the central portion that its only effects will be an irritation of the motor tracts. The latter localization will produce a progressive localized paralysis without preceding spasms. If it be cortical there will first be symptoms of irritation followed by those of destruction, i.e., spasms followed later by paralysis. The first irritative symptom will be tingling in a definite area, but this may be replaced by a numbness. This is a signal symptom, and indi-

icates the location of a growth much more clearly than the more pronounced symptoms of a later date. By reference to charts of the brain the growth can be easily localized when in the motor cortex, which is not true of tumor in other localities. As the growth increases in size the intensity of the irritation becomes greater, and spasms replace the paresthesias, but, however great the convulsive condition becomes, there is a precursory numbness or tingling which constitutes a sort of an aura, always of great localizing significance. The spasms at first are of slight intensity, and do not disturb consciousness, but are, from the first, followed by a temporary weakness which in recurring attacks becomes a temporary paralysis. Finally the slight initial spasms develop into tonic or clonic convulsions which recur at intervals, limited, or beginning upon one side of the body, and after several repetitions are succeeded by permanent paralysis. If a tumor is small, the spasm may occur again and again, and finally eventuate in a paralysis without a loss of consciousness at any stage. The order of the evolution of the condition is very important, since a general convulsion of sudden onset, and accompanied by loss of consciousness, is not a sign of tumor, unless the preceding symptoms have been present. If there is a permanent paresthesia, some ataxia with the paralysis, or a marked incoördination, or disturbance of the muscular sense, we are justified in localizing the growth in the motor zone behind the fissure of Rolando. Aphasia and agraphia would be produced, if the growth was in the lower portion of the motor zone, in front of the fissure. If the irritative symptoms were slight at first, increased slowly, and in paroxysms, and paralysis was late and rather slight, there would be a great probability that the tumor was in the meninges, impinging only on the cortex, or that the growth was subcortical. It might be very deep, if it was a large tumor. While focal epilepsy is usually a symptom, a tumor may yet be localized, as in the motor area, by a hemiplegia of very gradual onset, without other general signs of tumor. All these tumors produce an alteration in the deep reflexes, and one of the early signs of paralysis is a marked increase in the reflexes. Muscular twitching is a sign of impending paralysis, and at the same time of the location of the growth. The only atrophy in the muscles involved is one of disuse, and there are no electrical changes. Vasomotor disturbances and tachycardia have been symptoms.

Parietal Region. Destruction of this area gives rise to sensory symptoms in two ways, viz., directly and indirectly. Asternognosis is a direct result of injury to the superior parietal, and alexia and visual aphasia, if it is in the inferior lobe, if the left side is the one affected. Since gait is controlled by proper sensory information, growths in this region may produce ataxia, and since the cortical representation of the third nerve is near the angular

gyrus in the descending parietal, we may find a paralysis of this nerve. The indirect results may be numerous. If the growth pushes forward there will be spasms and paralysis, while if it dips downward it will irritate or cut off many of the tracts concerned in sensation. If it presses upon the tracts going to the posterior portion of the internal capsule from the rear portions of the brain, we shall find losses of both general and special sense. There will certainly be anesthesia and ataxia; if the optic tract is cut off we shall find bilateral homonymous hemianopsia, while a projection to the lower front portion will produce agraphia.

Tumors of the Temporal Lobe. The right temporal, in right-handed people, is considered to be a latent region, and a tumor will produce general symptoms only as a direct result, but by its pressure upon the brain stem and neighboring parts it may produce many confusing symptoms. If the pressure is backward there may be optic aphasia, while if it is downward there may be vertigo with forced movements, and a mixed type of aphasia. We may also see hemianesthesia, hemiplegia, and hemianopsia, and also ataxia. If it is on the left side in a right-handed person, it may injure the uncinate gyrus (same as hippocampal, and lies inward from the superior temporal) and directly produce loss of taste and smell, while if it affects the first and second temporal gyri there may be word-deafness, and the person affected will be unable to recall the names of persons and objects, and lose his power to understand spoken language. Amnesic aphasia results from this, but while deafness might theoretically be supposed to occur, it is a very rare result, since audition is probably represented in both hemispheres. Auditory hallucinations may, however, result.

Tumors of the Occipital Lobes. This is pre-eminently a visual center, and therefore both compression and destruction symptoms concern the sense of sight. Since the visual tracts are long, and a growth at many points may produce similar symptoms, such losses do not have a great localizing significance. If however, such losses are accompanied by sharp occipital pain extending into the neck, there is ground for localizing the cause in the occipital lobe. Even a small tumor will compress the tracts sufficiently to produce hemianopsia; and word-blindness will frequently accompany it. If on the left side there will also be mind-blindness, or psychic blindness, where with perfect sight the person fails to recognize a familiar object. These losses are as frequently the result of subcortical as cortical growths. Complete blindness may be produced by a bilateral lesion. We should assume that the growth was in the front, or had extended toward the front of the lobe, if, in addition to some of the before-mentioned losses, we should also find hemianesthesia, hemiataxia, and perhaps hemiplegia.

Tumors of the Sylvian Fissure, or the Island of Reil. By directly pressing upon the Island of Reil and the capsule, which lies under it, they may produce aphasia, and, if deep enough, give rise to all the symptoms of a tumor of the capsule itself. Indirectly tumors of the fissure of Sylvius may press upon the tip of the third frontal and produce motor aphasia, or, by invading the operculum (parts of the frontal and parietal which overlap the insula), paralyze the face, or so damage the inferior parietal, or superior temporal, that various forms of sensory aphasia are induced.

Tumors of the Central Parts of the Brain. These are unusual, and have no certain localizing symptoms. They simulate tumors of the frontal lobes in that the patients are immobile demented, with a great tendency to stupor, and they usually terminate in coma. If we find these symptoms with a hemiplegia, with a lesser degree of paralysis upon the other side, or a double hemiplegia with an absence of cranial nerve-involvement or anesthesia, it is an indication of a central lesion. The only focal symptom ascribed to them is a loss of the tactile sense, supposed to be located in the hippocampal gyrus.

Tumors of the Lenticular and Caudate Nuclei, and the Optic Thalamus. These are fairly common, and are principally marked by the general symptoms of tumor, and paralyzes resulting from encroachment upon the internal capsule. The functions of these ganglia are not sufficiently understood to make accurate localization possible, but diagnosis between growths in these bodies rests upon the fact that tumors of the lenticular and caudate nuclei are more apt to produce motor palsies, while those of the thalamus will give rise to a loss of the power to recognize the position of the limbs of the opposite side. Hence there will be an awkwardness of movement, the limbs will assume strange positions, and there will be athetoid movements. We may also see hemichorea. Some of these losses depend upon the disturbance of the cutaneous and muscle-sense. If the growth is in the pulvinar, there will be optic neuritis at an early date, while if it extends into the posterior part of the internal capsule, there will be hemianopsia, and, less often, hemianesthesia. If it projects into the lateral ventricle, there will be symptoms of distension. A tumor in the posterior portion of one thalamus invariably produces homonymous hemianopsia. The growth in the pulvinar produces a hemianopsia, which is distinguished from that resulting from a similar condition in the occipital lobe by the presence of a distinctive hemiopic pupillary reaction, or inactivity. A gradual hemiplegia, with twitchings and jaetitation of the limbs preceding it, is a sign of encroachment on the motor tracts.

Tumors of the Corpora Quadrigemina. These growths are rarely limited to these bodies, but usually involve the crus and

pineal gland, or "vice versa." Since these bodies are the primary centers of sight and hearing, and through the crus, in addition, pass both sensory and motor tracts, the symptoms are very wide in possible range. The corpora quadrigemina are the seats of the coordination of the movements of the eyes, and therefore all reflex motions of the eyes are interfered with. Growths usually are sufficiently large to compress the oculo-motor fibres, and diplopia will result. The eyes cannot follow a light, or gaze steadily at a page, and therefore such ocular losses, combined with a reeling gait and the general symptoms of tumor, will localize it in the corpora quadrigemina. If the crus is also compressed by a growth in this locality, there will be a "superior alternating hemiplegia," that is, there will be an oculo-motor paralysis of one side, accompanied by a paralysis of the extremities of the other side. This is the syndrome of Weber, and is also said to be caused by a tumor (tuberculous or syphilitic) on the inferior and inner side of the crus, and involves also the face, tongue and limbs of the opposite side. In tumors of the quadrigemina there is loss of the pupillary reflex, and nystagmus is present.

Tumor of the Crus. If a tumor occurs here, we shall typically have a third nerve palsy of one side (which indicates the side upon which is the tumor), and a hemiplegia of the other. Extension of the tumor may induce the paralyses just mentioned, and if it compresses the optic tracts there will be bilateral hemianopsia. An intention tremor is also found, and also one similar to paralysis agitans.

Tumors of the Pons. A tumor may be so located as to give rise to few symptoms, until it attains a considerable size. The usual symptoms are a combination of hemiplegia of an alternating type, and cranial nerve palsies. Some combination is true in 85% of the cases, but some are solely hemiplegic, and a few produce lesions of the cranial nerves alone. Bilateral symptoms are more common than in lesions of the higher levels. The paralyses of the limbs will be of the upper-neuron type, i.e., increased reflexes, slight atrophy, and unchanged electrical reactions, while those of the cranial-nerves will be of the lower-neuron type, i.e., lost reflexes, decided atrophies, and the electrical reaction of degeneration. Rulably a tumor of the upper part of the pons will paralyze the third and fifth nerves, producing oculo-motor palsy, dilatation of the pupils, ptosis, tingling, pain and anesthesia of the face, sometimes ulceration of the cornea, and, at times, grinding of the teeth in sleep. A lower localization produces a paralysis of the sixth nerve, which allows the eyeball to rotate inwards. Below this it will involve the seventh nerve, and facial palsy will result, while an impairment of the eighth will cause deafness. Conjugate deviation of the eyes is toward the side opposite the lesion, just contrary to the condition entailed by a cerebral lesion (the

eyes look at the lesion in cerebral destructive lesions, and away from it in pontile destruction), is sometimes an accompaniment of facial palsy, and under such circumstances the face will be palsied on one side, and the eyes will deviate to the other. Tumors in this locality are liable to exert pressure upon the middle lobe of the cerebellum, and under such circumstances there will be a staggering gait, usually deviating toward the side opposite the tumor, forced movements, and general unilateral, or alternating convulsive movements. These tumors of the pons uniformly produce a loss of knee-jerk, and the control over the bladder and rectum is frequently impaired.

Tumors of the Medulla. Here also there is a combination of cranial-nerve symptoms and hemiplegia. Irritative symptoms of the ninth, tenth and eleventh nerves are most usual, and therefore there are difficulty in swallowing, irregular action of the heart and of respiration, and vomiting, and sometimes retraction of the head and neck. The twelfth nerve is least frequently affected. There may be numbness and tingling, and if there is pressure on the cerebellum, there may be cerebellar ataxia, or the limbs may be paralyzed upon one or both sides, in both motion and sensation. The termination is usually in convulsions.

Tumors of the Base. They often give rise to symptoms referred to the lobes which they underlie, but continuous pressure often produces a perforation of the bones, and they protrude externally, or into the cavities in their neighborhood. They often invade the orbit, nose, mouth, antrum and tympanum, and their presence is indicated by discharge of brain-tissue, blood, or the structures of the tumor.

Tumors of the Cerebellum. On account of its confined position between the base of the skull and the tentorium, any tumor, however small, will produce symptoms resulting from pressure, and a tumor in the middle lobe will give rise to focal symptoms of a very distinctive character, but a tumor located in the lateral lobes may attain a very considerable size, and produce no symptoms of a focal character. The lateral lobes are latent zones, and all symptoms of a focal nature are the result of pressure upon contiguous parts, and are therefore those which are called indirect. General symptoms are developed earlier than in tumors of the cerebrum, and are the following: acute headache in the frontal or occipital region with pain extending down the neck and into the back, stiffness of the neck, and optic neuritis which is very early and intense, and more frequent than in tumors of other localities. There are general and unilateral convulsions, and opisthotonos, and muscular rigidity. If located in the middle lobe there is always cerebellar ataxia, which is a staggering gait with an irregularity of the steps, and a swaying of the body as if the person were intoxicated. This is attended by a vertigo which comes on early

and persists, but which is relieved when the patient lies down. The patient rarely falls, and actual ataxia of the extremities is more rare. Early and continuous vomiting is also a symptom. The early appearance of cranial nerve-symptoms, such as paralysis of the face, deafness, disturbance of swallowing, and symptoms indicating an implication of the vagus, indicates that the tumor lies near the medulla and pons, i.e., on the inferior surface of the cerebellum. Symptoms of irritation, like convulsive tic, often precede the paralysis. Nystagmus is not an unusual symptom. The knee-jerks are usually exaggerated in cerebellar tumor, but cases have been seen where they have been abolished. If the nerves of one side are affected, it indicates that the pressure is rather upon nerve-roots than upon the medulla, and so localizes the growth as upon that side.

DIAGNOSIS. There are three phases in which the diagnosis of tumor must be considered. Is there a tumor? Where is the tumor? What is its nature? We should diagnose a tumor from the presence of the following symptoms, viz.: a constant and quite severe headache, vomiting of a cerebral character, double optic neuritis, retardation of the pulse, generalized spasms, somnolence, mental dullness, and vertigo. But more briefly, we may reason that while headache is common to many diseases, and conditions, if papillitis, or double-choked disc is also present, the cause of the headache is almost certainly tumor. Even if a person has headache with papillitis, the presence of an accompanying chlorosis should cause us to be guarded in making a diagnosis. If however we can add to headache and choked disc, projectile vomiting and vertigo, the diagnosis is certain.

DIAGNOSIS OF SITUATION. For this element in the diagnosis it is necessary to carefully separate the general from the focal symptoms, and to assure ourselves of the order of their appearance. When the general symptoms are pronounced, and the focal symptoms are indefinite, or altogether wanting, there is a strong probability that the growth is in the frontal or temporal region of the right side, or is in one of the lateral lobes of the cerebellum. The combination of the symptoms may be as characteristic of their location as is their order, or mode of extension. This is especially true of tumors of the crus, and the corpora quadrigemina. When a tumor is in such a position that it exerts pressure upon the large vessels, or interferes with the general circulation, it gives rise to symptoms indicative of thrombosis. The symptoms may be direct, or indirect.

NATURE OF THE GROWTH. We now come to the third factor in making a diagnosis. When we are so fortunate as to be able to localize it, we have, from that fact, some grounds for a surmise as to its nature. Tubercle and glioma are more liable to be found in the pons and cerebellum, while sarcoma is frequent at the base,

and outside of it on the walls. Syphiloma is common in the pons, basilar area, and the cortex, but is rare in the white-matter of the brain and the cerebellum. Fibroma and glioma are interstitial growths, and favor the deep structures, while tumors of the ventricles and meninges are liable to be sarcomata. The stage of life also bears upon this subject, since in childhood we are apt to find tubercle, glioma and sarcoma, while adults are more liable to develop the harder growths and syphiloma, while carcinoma is commonly confined to an advanced period of life. The clinical history is another element in the formation of our opinion. A rapid onset followed by a period of stasis, especially if the patient be in the first half of life, indicates that the growth is tubercle. Glioma and sarcoma are steady and insidious in their rate of progress, while syphilis is rapid in onset and course. Apoplectic seizures are an indication that the growth is a glioma, since such tumors are most frequently subject to hemorrhage. The condition of other parts of the body is a strong indication of the variety of morbid process going on in the brain. Syphilis, cancer of the breast, or of other regions, actinomyces of the jaw, and hydatids of the liver strongly indicate that a growth of similar nature is in the brain.

DIFFERENTIAL DIAGNOSIS. Nephritis in any form may be distinguished by the history, by examination of the urine, and the state of the heart and arteries.

Lead intoxication presents the history of a favoring occupation, repeated attacks of colic, and an obstinate constipation. Failing such a history we should discover a blue line about the edge of the gums, signs of lead in the urine, a tremor of the extremities, and possibly wrist-drop.

Hypermetropia in anemic girls will sometimes give rise to a severe headache, occasionally with vomiting, and a slight degree of choked disc may be found. A differentiation cannot always be made, but generally it is cleared up, from the fact that the headache and the vomiting are relieved by glasses, and the diagnosis is made certain if the anemia can be relieved.

Profound Anemia may give rise to all the general symptoms of tumor, but if the anemia is benefitted the symptoms abate, and at any rate focal symptoms do not appear.

Amenorrhea may produce all the symptoms described in speaking of anemia, and no anemia may be present, but if the menstrual function be restored, the diagnosis is at once cleared up.

Hydrocephalus of a chronic form, however produced, develops pressure symptoms resembling those of tumor, and can only be differentiated by the focal symptoms. The diagnostic point is the order in which the symptoms appear. The results are a spastic paralysis, but it develops without antecedent spasms. It is moreover bilateral, and the lower limbs are affected more

than the upper, and there is incoördination of the hands. As the pressure increases, the crus and pons may be displaced, and the stretching of the cranial nerves in an irregular way will produce unusual symptoms. The cranial nerve symptoms, with a paralysis of the lower limbs, would lead one to think of a tumor of the base.

Chronic Cerebritis or Encephalitis, may simulate a tumor very closely, but the cases are very rare.

Acute Encephalitis gives rise to an optic neuritis, but it differs in being an acute febrile disorder, and differs also in its later history, in the fact that it is usually fatal in a few weeks, or terminates in recovery in a somewhat longer time.

Meningitis, acute or chronic, is comparatively rapid in its development, as compared with the months which must elapse in the development of the most quickly growing tumor. Meningitis causes a neuro-retinitis, while a tumor produces choked disc. In meningitis the paralyzes are bilateral, while they are unilateral in tumor, unless it is multiple. The paralyzes of meningitis are slighter and more variable in their duration, and there is fever, marked variation in the size of the pupils, a slow and hard pulse, constipation, retraction of the abdomen, and vasomotor symptoms. Pus will be found in the spinal canal, and, post-mortem, tubercles are more often found on the choroid following meningitis than after a tuberculous tumor. Certain diagnosis can only be made in the tuberculous forms when the case of meningitis is one of those which develops rapidly, is complicated by hydrocephalus, and is fatal in three or four weeks. In general it may be said that the headache of meningitis is more continuous and severe, that a neuro-retinitis is less rapid and intense than the choked disc of tumor, and that a general subsidence of symptoms with recovery would indicate a meningitis rather than a tumor. If the meningitis is at the base, certain diagnosis is impossible, since a tumor is often complicated by a meningitis. A chronic meningitis of the base especially imitates a tumor of the sella turcica, since the same cranial nerves will be implicated in both conditions, but the meningitis produces a descending optic neuritis without coincident symptoms of an increase of intracranial pressure. When in young children, a diagnosis must be made between tuberculous meningitis and tumor, we should lean toward the former if there was blindness with a premature closure of the fontanelles, while an enlargement of the head would point to tumor.

Abscess. A latent cerebral abscess presents all the symptoms of tumor, which in fact it is, but with a better prognosis. Diagnosis must often be made from its etiology. Abscess occurs after severe injuries, or suppurative conditions of the ear, nasal cavities, orbit, antrum or cranial bones. After a severe injury of the

skull, we may find either a tumor or an abscess, and it is never proper to assume it is a tumor if a suppurative focus is present. Unless the abscess is encapsulated, and still uncomplicated by cerebritis, meningitis, or sinus phlebitis, the symptoms appear in more rapid succession, and with greater severity than in tumor, and with fever. After a severe range of symptoms there may be a period of latency, after which there is a new exacerbation, which leads to a fatal issue. Tumors are marked by a constant accession of new symptoms, which is not true of abscess. While the headache is more severe and paroxysmal in tumor, a unilateral localization is rather a sign of abscess. Optic neuritis is more common with tumor, and mental changes are more gradual and constant, and the local symptoms appear more slowly. The temperature is normal, or subnormal, in both, but if we find sudden fluctuations of temperature with sweat, abscess is the more probable cause. Marked emaciation is more often a characteristic of abscess, but it occurs in an unaccountable manner in some tumors. Intra-cerebral hemorrhages are a common incident in tumor, while meningitis is the usual complication of abscess. Symptoms of sinus phlebitis indicate abscess. A duration of one to two years, with no accession of new symptoms, indicates abscess. After such a period there can be no doubt, unless the tumor is situated in some latent area, and so has attained a considerable size without marked symptoms. Hemorrhage would then differentiate, unless it could be explained by an injury, or some degeneration of the arteries.

Cerebral Hemorrhage or Softening. The arterial system should be examined to furnish a reason for the attack, and also the previous history of the patient, and the symptoms succeeding the hemorrhage. If a hemorrhage has resulted from arterial degeneration, the symptoms will decrease after a time, and there will be no optic neuritis, but if the hemorrhage has arisen from the rupture of vessels in a tumor, the symptoms will show a steady increase after the hemorrhage, there will be headache, convulsions, and optic neuritis.

General Paresis. Ordinary cases show no similarity, but some doubt may arise when it begins with persistent headache, and paralytic and irritative symptoms are present, whose exact nature cannot be identified without accessory symptoms. Tumor of the medulla may present a quite similar symptomatology. Choked disc does not occur in paresis, but it is replaced by pupillary rigidity. The mental state of tumor is stuporous, while that of paresis is some grade of grandiose delusions, or a definite melancholy, going on to dementia. Paresis has apoplectiform attacks, which resemble cortical epilepsy from tumor, but the result is a decrease in intelligence, rather than the paralysis occurring in tumor. Tumor at the base may be marked by tremor and dis-

orders of speech, but such growths do not produce the combination of a tremor of the lips, upon strong retraction, with slurring speech.

Epilepsy has convulsions similar to those occurring in the history of tumor, but with differences. The convulsions from tumor have a clonic stage followed by the tonic, while the opposite order obtains in epilepsy. The convulsion of epilepsy lasts for two or three minutes only, while that of cortical or Jacksonian epilepsy (the variety found in tumor) lasts many minutes, and it may be hours. An epileptic convulsion is terminated by a deep sleep of a half-hour, to one or two hours duration, while that is replaced in focal epilepsy by a slight state of coma. The convulsion of the epileptic may leave him mentally dull for some hours, but he does not suffer from the severe headache, the projectile vomiting, or vertigo, neither does he show choked disc.

Migraine. This may have a severe headache followed by a choking of the disc, in rare cases, and the pulse may be retarded, but the condition is a periodic one. Some cases of migraine become constant after a time, but the disc will not show choking under such conditions, and further and distinctively, the disease presents the same group of symptoms without much variation, while a case of tumor is notable from the constant accretion of new symptoms.

Disseminated Sclerosis. Tumor may present such symptoms that the diagnosis is doubtful if it be located at the base, since a tumor in the cerebellum, pons or corpora quadrigemina may give rise to nystagmus, tremor, headache, vertigo, and an uncertain gait. It will be especially confusing if the case is one in which choked disc has reached its final stage, and produced optic atrophy, which is a symptom of disseminated sclerosis. The points of difference are these: The tremor is just as often passive as it is intention in type, and if intention it is more indefinite, and is combined with ataxia, while that of disseminated sclerosis is combined with spasticity. We rarely find a case of disseminated sclerosis which does not develop spinal symptoms, while in it we do not find stupor, retardation of the pulse, vomiting, aphasia, cortical epilepsy and spasms.

Hysteria may so resemble the symptoms of tumor, that the diagnosis must depend upon exclusion. The headache of hysteria may be as violent as that of tumor, but it is characteristically aggravated if attention is directed to it. This is also true of neurasthenia. If optic neuritis is developed, its presence will clear up the diagnosis, since this has never been seen in hysteria. It should be remembered that the presence of a tumor may induce hysteria in a favorable subject.

Intra-cranial Aneurism. This is, to all intents and purposes a tumor. It may sometimes be diagnosed from the presence of a

bruit, or the bone may become so eroded by its pressure that pulsation may be made out, or it may invade one of the sinuses, giving the symptoms of sinus thrombosis. It should be thought to be a possibility if there has been syphilis, general arterial degeneration, or an injury to the skull.

Alcoholism. Chronic alcoholism may produce many of the symptoms suggesting the presence of a tumor. There may be headache, apoplectiform seizures, vomiting, and even optic neuritis. This latter condition furnishes a ground for differentiation, since the optic neuritis from alcohol has a central scotoma for colors. Usually there are the characteristics of gastritis, diarrhea, and a general tremor of the body, limbs, lips and tongue without any in the face.

Multiple Neuritis. This, whether from alcohol or a cachexia, may produce an optic neuritis, but if the limbs are weakened by a tumor, the deep reflexes are exaggerated in place of the diminution or loss found in multiple neuritis.

Prognosis. This depends upon the age of the patient, the kind of growth present, and its location. The prognosis for duration of life, and return to health, is better in the child than in the adult. The compression symptoms are not so severe, since the cranium is more elastic, and the sutures separate more easily, and children are more liable to be affected by tubercle, which may be operable, and is thought to be more liable to remissions, and sometimes spontaneously absorbs. The average length of life of a patient giving signs of the presence of a tumor is from two to four years, but some cases die in a few months, and some have lived as many as ten, or even fourteen years. A sudden fatality is always imminent, but remissions often occur, and have been known to endure as long as eight years, and many cases have a period of latency, after which there is renewed growth. Another method of spontaneous relief of a temporary character is by the perforation of the bones by the growth. When this occurs, there is a discharge of the ventricular fluid, often through the nose, which temporarily relieves the intra-cranial pressure.

Different varieties of growth give rise to some degree of difference in the prognosis. Solitary tubercle, glioma and the hard varieties of sarcoma are of slow growth, cholesteomata and cavernous angiomas are especially chronic. The soft sarcomata, and carcinomata are rapidly fatal. Taken as a whole the prognosis is best in syphiloma, but they are prone to return. Echinococcus cysts, and cavernous angiomas, and solitary tubercle, are of relatively good prognosis. Diffuse tubercle (as opposed to the solitary form) and syphilis may have a bad prognosis, since they are apt to be complicated by meningitis. Any form of tubercle and carcinomata has a bad prognosis, since it is usually secondary to like conditions in other parts of the body. Any growth which

is hemorrhagic has a grave prognosis, since there is a great liability to apoplexy. This hemorrhage may be into the brain about the growth, in which case it has the usual prognosis, but it may be into the growth, and affect the brain only by increased compression. In this latter case the initial fall of temperature peculiar to cerebral hemorrhage is said to be absent. Spasms indicate cortical irritation, and paralysis destruction of the motor cortex, or of the motor paths.

Favorable symptoms are a subsidence of the headache, and a recession of the optic neuritis, and if the optic neuritis develops slowly, it indicates a like rate of development in the growth. Symptoms of bad omen are a stuporous mental condition, convulsions, severe vomiting (both from its causation and also its effect upon nutrition), a rapidly developing and severe grade of optic neuritis, apoplectiform seizures, and, above all others, bulbar symptoms. Aside from syphilitic tumors, which are sometimes amenable to remedies, the prognosis depends upon the possibility of removal of the growth by surgical means, and a tabulation of recent statistics indicates that there is a possibility in from five to nine per cent. Practically the only tumors capable of removal are those which are cortical, or closely subcortical, in the motor zone. This arises from the fact that they are accessible, and give rise at an early date to localizing symptoms, and can be removed before they attain a great size. Large tumors have been successfully removed, but the probability of success is less. If small they may be removed, even if of the diffuse variety, but when large they are operable only if they have a capsule, or are in some way circumscribed. Multiple tumors are always inoperable. In some cases this rule is inapplicable, since some diffuse tuberculous and syphilitic growths have been excised with a favorable result. Aside from growths of the motor zone we should be justified in operating over the third frontal on the left side, if in addition to the general symptoms of tumor, there had been from the beginning some degree of aphasia. Under the same general conditions we might operate over the occipital lobe, if there had been an early and constant hemianopsia, and over the temporal if the loss had been one of audition. Cerebellar tumors have been successfully operated, but it is very exceptional. Forty-eight cerebellar tumors were operated upon with 20 deaths, 16 recurred, and one was apparently cured. Cerebellar cysts have a good prognosis. Tubercle is operable at times, even when complicated by meningitis. Basilar, pontile, and medullary regions are outside the possible field. Carcinoma is the least operable of all. Psammomata, cholesteomata, angioma, dermoid cyst, and the parasitic form are operable if they can be localized. Sarcomata can usually be enucleated, tubercles and fibroid tumors shelled out, but gliomata are infiltrating, and cannot be completely removed, and

are liable to recur from the remnants. Cysts and old abscesses are readily drained. If the growth is in the lateral lobes, localizing symptoms are absent, and if in the middle lobe it is inaccessible.

Death results from cachexia, induced by pressure and consequent inhibition of cerebral functions, or from hemorrhage, convulsions, exhaustion, infection, bulbar symptoms, or intercurrent maladies.

TREATMENT. If the growth is syphilitic a mixed treatment sometimes produces brilliant results, and the same effect has been obtained in tumors which were later discovered to be sarcomata, but in both cases there is a large probability of rapid recurrence. If the growth is tuberculous it is possible that iodoform may produce as favorable a result as it sometimes does in growths upon the peritoneum and meninges. The method employed by Dr. Sidney F. Wilcox of New York in those cases is worthy of a trial upon tumors. Shave the head, and once a day rub in a liberal quantity of the following preparation: Iodoform, 2 drams, Ether, 2½ ounces, Sweet Oil to make 8 ounces. If the case is an operable one, an early operation is to be advised, since delay increases the resulting damage to the brain, even if it does not render operation impossible. This possibility has already been defined.

THERAPEUTICS. In inoperable cases we have slight grounds for hope from remedies, but the following have been advised: The results from the administration of Thuja Tr. have been so striking that it is possible that we may find benefit from its use. Arnica, Arsenicum, Arsenicum iod., Baryta carb., Belladonna, Calcarea carb., Carboic acid, Conium, Crocus, Gallium in substantial doses, Graphites, Hydrastis, Kali hyd., Lachesis, Nitric acid, Phosphorus, Sepia, Silicea.

When the pain in the head is excessive, it will be found necessary to invoke some one of the analgesics, and morphine may be found to be the only one of much utility.

APHASIA.

DEFINITION. Aphasia, in its most complete form, indicates a condition where a person is unable to convey his wishes or intentions to others by means of speech, writing, or signs, his hearing and sight being at the same time unimpaired, and the muscles necessary for the accomplishment of his object being intact. There are many degrees and varieties of this loss to which definite names have been attached, and are commonly accepted. Two great divisions have been recognized, viz.: Sensory and Motor Aphasia. If the aphasia depends upon an injury which cuts off the reception of auditory impressions, the result will be termed Auditory Aphasia, Word-deafness or Amnesia. If it relates to the visual impressions we shall find Visual Aphasia, Word-blind-

ness or Alexia. If the loss is so extensive that the patient is unable to frame gestures by which to express his interpretations or desires, this condition has been termed Amimia. If in addition to faults of expression he can form no impression of the uses of objects by the tests of smell, touch, feeling, etc., he suffers from Apraxia, or Mind-blindness. This group comprises the sensory aphasiae. If the perceptions are intact, but the power of audible speech is lost, the fault is termed Motor Aphasia, but if it is solely of the power of communication by writing it is Agraphia. Losses of all kinds may not be in the power of expression by certain devices, but in a faulty use of powers yet present. He may still be able to speak, but uses wrong words, that is Paraphasia; if wrong words are written the fault is Paragraphia, while if wrong gestures are used it is Paramimia. If the patient fails in one only of these particulars the case is one of Pure Aphasia, but this is uncommon, and a failure to speak often entails an equal inability to write, or there is some other combination of defects. These losses have been considered to depend upon lesions of certain specialized centers. Other cases have been observed where such centers were intact, but the tracts between these centers were impaired, and to these cases the name of Conduction Aphasia has been applied. On this difference of site has been based a division into Cortical and Subcortical Aphasiae.

DIFFERENTIAL DIAGNOSIS. The only conditions from which aphasia must be differentiated are Aphonia, Anarthria, Dysarthria, and Mutism from mental disease.

PHYSIOLOGY. Until very recently the opinion has been held that impressions from the outer world were stored up in specific and definitely limited areas of the cortex of the brain. Although belief in this classical theory has been seriously shaken, it will be stated at some length, since such a physical localization of functions is an aid to a comprehension of the condition. It is much easier to reason from the tangible to the intangible than vice versa. Speech is receptive and emissive; we hear, and see, and feel, and smell and touch, on the receptive side, and at a little later period perceive that the persons about us gain their desires by making certain gestures and sounds, and forming certain characters. Some persons gain more of their knowledge through the ear than through the eye, and are Auditatives, while others learn more from sight, and are Visuals. By whichever avenue our knowledge is gained, there is a combination of the two methods in some proportion. It has generally been assumed that sounds, as sounds, are stored up in the first temporal convolution, but that when they assume the dignity of word-concepts they are preserved in the cortex of the supra-marginal gyrus, which is that part of the descending parietal convolution which lies about the upper extremity of the Sylvian fissure ("pli courbe" of Marie).

Objects which are seen are mentally appreciated in the cuneus, but the memories of these objects, as sight-concepts, are preserved in the angular gyrus, i.e., that portion of the occipital lobe which lies about the posterior extremity of the superior temporal fissure. Memories of muscular impressions have been thought to be stored up in a cortical area of the descending parietal lobe lying just above the supra-marginal gyrus. The center for the production of speech is in the posterior part of the lowest (third) frontal convolution, and the anterior part of the Insula (Broca's convolution). The center for writing is in the posterior portion of the second frontal convolution, part of Broca's area, and the center for stimulation to voluntary speech lies in the cortex of the anterior portion of the frontal lobes. The main avenues of association-tracts between the receptive centers for sound, sight, and touch, and the emissive centers for speech and writing, are through the subcortical portion of the insula. Receptivity is at first bilateral, and therefore children before the age of seven are rarely aphasics from unilateral cerebral injuries, but, at about that age, the function becomes strongly localized upon the left side in right-handed people. These centers are put into association with one another, so as to act in correlation by subcortical tracts, and some centers cannot act independently of one another, since the visual seems very dependent upon the auditory, and the graphic upon the motor speech; this explains the frequency of mixed forms of aphasia. Physiologically the development of the faculty of emissive speech is simply the process of the elaboration of these conduction and association tracts. By the end of the first year of life the child has begun to appreciate the inherent differences in objects, and that people gain them by the use of sounds, and has grasped a rudimentary knowledge of them. By the end of the second year he has practiced these sounds to such an extent that he has a partial command of them, and an amplification of this process along the same lines is all that is requisite for the attainment of perfect speech. Teaching the hand to copy forms, which he has learned to be the analogues of sounds, completes his education in emissive speech. The progress in education will be by eye and ear, and will differ in degree in different people, but further education is a greater accumulation of such visual or sound impressions, and such a degree of inward repetition of these stored-up impressions that they will be available on demand. This repetition is usually by speaking them to ourselves, but in strong visuals will be in throwing up to the mind the appearance on the page, or as the object appears in the world. Afterward this is only done when an object or word is unfamiliar, and, from his greater store, the educated man halts less in speech or writing than the uneducated one. If we desire to speak, we conceive of the general concept in the anterior portion of the cortex of the frontal lobe.

We seek the desired word in the word-concept center in the supra-marginal gyrus, and sound it to ourselves by setting into action the conduction-tracts running through the subcortical portion of the Island of Reil to the motor-speech center in the posterior portion of the third frontal convolution. We test this sound by stimulating the tract leading to the center for sound memories in the first temporal convolution. Having satisfied ourselves that the sound is correct, the motor-speech center is again stimulated, and an impulse is sent downward through a tract near the knee of the internal capsule (the aphasic fasciculus of Raymond and Artaud) to the nuclei in the medulla, and the requisite muscles for voice-production are set into action.

For writing, the concept is thrown up in the visual concept-center (angular gyrus). It is tested in the cuneus by stimulation of the tracts to that area of the cortex. If the picture is found to be correct, impulses are then sent along the tracts to the graphic-center, which is in the posterior portion of the cortex of the second frontal convolution, whence impulses are sent down through the tract posterior to the knee of the internal capsule to the proper muscles of the hand and arm. For the sake of simplicity we have assumed that all concepts are single, and move along a single line, from the conception of an idea to its final evolution in speech or writing, but such a condition is rare. The process is probably a most intricate combination. We invoke every variety of memory of the subject of the concept; its appearance, its feeling, and form, its picture, and its name, so that in the classical, and perhaps outlawed conception of aphasia, the complex character of the process has always been recognized. The essential difference between an educated and an uneducated person, rests upon the ability of the former to act directly from the concept-center in the frontal lobe without the laborious process of testing the proper form of desired concepts by interrogation of the specialized centers on memory. He also has a more elaborate system of association-tracts, and a simple suggestion from the outside world calls up a much wider range of associated concepts.

This theory of the localization of speech, both receptive and emissive, in specific centers, has recently been attacked by Marie, of Paris, in a most definite manner, and has already seriously shaken the classical hypothesis. His success results largely from the fact that it had been previously noted that post-mortem findings had often failed to confirm a clinical diagnosis. Dercum states (from Fraenkel and Onuf) that out of one hundred and four cases of motor aphasia only seven showed a lesion limited to Broca's convolution. In five of these the aphasia was transient, in two only was it persistent. Dejerine in twenty-five years was unable to find a single case, but based his belief in this localization upon two cases of Bernheim and Ladame. Marie disproved

Bernheim's case, and Ladame announced that the study of his case was not complete. Marie based his conclusions upon the results of fifty autopsies at the Bicêtre. He says that Wernicke's assumption of a specific auditory, olfactory, visual, and motor image-center lacks foundation, and that Broca's convolution, as a defined motor-speech center, plays no especial role in the function of language, and rested upon the findings in only two autopsies. He acknowledges, that in softening, for instance, which is followed by aphasia, Broca's convolution may be found to be degenerated, but he says that this is an accidental concomitant only of the actual condition causing the aphasia, and that this actual causative factor is mental deterioration. In the so-called Wernicke's (sensory) aphasia the patient can talk, at times he presents the picture of a perfect jargon, aphasia, or paraphasia; he may understand what is said to him, but understands it imperfectly, and, as stated, Marie believes that this is not due to a loss of verbal images, but to a generalized intellectual impairment. In Broca's (motor) aphasia the patient cannot talk at all, and yet shows an intellectual incapacity to write, read, etc. This seems to Marie to indicate that a motor aphasia represents a sensory (Wernicke's) aphasia, plus a loss of the power of speaking, (an anarthria). In the new formula therefore, a sensory aphasia plus anarthria equals motor aphasia. Marie localizes the true center of language, the destruction of which causes a sensory aphasia, in the supra-marginal gyrus, the "*pli courbe*," in the base of the temporal convolutions, and in the fibres coming from this region. This is the region considered by Flechsig to be peculiarly an association area. Marie, moreover, localizes lesions causing motor aphasia in the sensory aphasia area, or in the fibres coming from this area, combined with a further lesion in the area known to be affected when anarthria is present (lenticular nucleus). He says also, that we can no longer affirm that certain aphasias are cortical while others are subcortical, but that both zones are always implicated. We can much better divide them into the intrinsic and extrinsic. They are intrinsic where the zone of language is directly affected, and extrinsic when the lesion is in the lingual and fusiform lobes (pure alexia, or word-blindness), or in the zone of the lenticular nucleus (pure anarthria; pure motor aphasia). The zone of Wernicke is not only a sensory, but also an intellectual center, and every lesion of this center produces a proportionate disturbance of the power of speech, of the comprehension of spoken words, of reading and writing, and a loss of certain ideas of a didactic order. Anarthria is clinically characterized by the loss of the power of speech, with the retention of the comprehension of words in reading or writing. This is produced by a lesion in the zone of the lenticular nucleus which interferes with the coördination of the muscles necessary to the articulation of words, without

any true paralysis of the muscles of phonation being present. Under the influence of Marie's statements, Dercum re-examined fourteen cases of aphasia, and found that what words they could utter differed very greatly from the speech of normal persons. He cites Marie in saying that anarthria may be due to an inharmonious action of the various organs used in articulation, viz., larynx, tongue, lips, and the muscles of expiration. He concludes that Marie's contention that aphasia is a unit is well borne out by the facts, but he thinks that it has not yet been proved that aphasia is due to involvement of the zone of Wernicke, or that all of the motor-speech phenomena are due to an involvement of the zone of the lenticular nucleus, and that a lesion of the third left frontal convolution has nothing to do with motor aphasia.

PATHOLOGY. However much, or little, centers for speech are localized, it is a clinical fact that the destruction of certain tracts and areas in the brain will interfere with the perfection of speech, and if the lesion is sufficiently extensive, the result will be a total abolition of it. This is the result of pressure upon the areas which have been referred to, or of deprivation of the supply of blood to the parts. The depression of a corresponding part of the skull, or a subdural or meningeal hemorrhage, is a common cause of pressure paralysis, while a cerebral hemorrhage, or thrombosis, or embolism, will cause softening. Arterial spasm may be so prolonged as to cause, or infections may produce, emboli in the meninges. Shocks to the emotions may have caused the spasm of the arteries, and infections or toxins may act directly upon the cells of the cortex or ganglia. It is a very usual result of thrombosis, since this customarily occurs in the larger arteries, and a stoppage of circulation in the anterior or posterior cerebrals will cause some form of aphasia, and these are the arteries of election for thrombosis. Motor if in the anterior, and auditory and visual if in the posterior. Fully two-thirds of the cortex of the left hemisphere are concerned in speech, and the upper auditory center is most important, since injuries of this area almost universally implicate the visual areas also.

SYMPTOMATOLOGY. If the auditory center is affected, Word-deafness ensues, and the person cannot understand spoken speech, but if the type is pure, he can read writing and printing. He is also paraphasic, because, as has been said, we repeat to ourselves what we are to emit, and he cannot hear himself test his premeditated expressions. He may be paragraphic also, unless he is a strong visual. He cannot repeat from dictation, because he has lost sound memories, but he can copy writing, because he still preserves his sight, and muscular memories. He cannot write from dictation, but he may be able to sing. If the lesion is bilateral, he may have lost, or impaired memories for sounds, and will

have lasting word-deafness (aphasia,) and in such cases his mind is impaired.

Lesions of the Visual-center. Word-blindness results, and he cannot write (agraphia), since he cannot remember the appearance of written signs. He cannot understand writing for the same reason, but, of course, can understand speech. He can repeat on request, and he can write, because that is mechanical, and does not demand interpretation from the visual center. Mental impairment is slight. If the lesion is extensive, he will suffer a loss of vision in the right half of each eye (homonymous hemianopsia).

Lesion of the Motor-speech Center. The patient will be dumb. He can understand spoken speech, and written, partly, but he cannot articulate correctly, and often is incapable of writing. He cannot repeat from dictation, but can generally copy from writing. He may be able to write from dictation, and he may not be able to make his wants known by signs, or his power in this direction may be imperfect.

Conduction Aphasia. The simplest form is where the tracts running between the Island of Reil, and connecting the auditory and the motor-speech areas, are severed. Such persons can speak and write, but inasmuch as in speaking and writing we continually correct ourselves by inward repetition, and here the auditory center has been cut off, he will use wrong words both in speaking (paraphasia), and in writing (paragraphia). He can understand spoken and written speech, but in repeating from dictation he is paraphasic. If he is writing he will do it correctly, but if from dictation, he is liable to be paragraphic.

PROGNOSIS. Time alone can decide whether the aphasia from a shock to the brain is functional and temporary, or organic, and, to some degree at least, permanent. This cannot be decided in less than a week. Word-deafness is of a better prognosis than motor aphasia. Substitution of function from the uninjured hemisphere is rare after the seventh year, but before this time perfect recovery is the rule, unless the mental powers of the brain have been seriously compromised. The mind is more apt to be affected after an auditory loss. The oldest impressions are more apt to be the first regained, and the loss is most marked in proper names, and next in nouns.

DIAGNOSIS. It is first necessary to determine if a patient sees and hears, and that the muscles of phonation are not paralyzed. Then, avoiding gestures, ask him to perform simple motions, and next to name objects about him. Next see if he recognizes by sight objects of which he understands the names. Observe whether he is paraphasic, which will be shown by his use of a wrong word, and while he still evidently appreciates the actual character of an object. Slowness in answers will show partial

loss. Find whether he can repeat words pronounced to him, or can read and write. He should be tested in spontaneous writing, copying, and from dictation. If apparently unable to read, see if he can read a simple request. Examine into powers of continuous thought for the mental defect which will be present. This will be evidenced by a correct answer to one or two tests along the same line, and subsequent repetition of failure upon further investigation.

DIFFERENTIAL DIAGNOSIS. Aphonia is the condition where all language is perfectly comprehended, but speech is impossible because the vocal cords are paralyzed, as will be evident upon laryngoscopic examination.

Anarthria is a condition where tests will show that intellect and comprehension of all forms of expression are perfect, but there is a total paralysis of the vocal apparatus.

Dysarthria is a similar condition as to the comprehension of speech, but language is poorly enunciated, varying according to the different degree of involvement of particular sets of muscles. Bulbar speech is an example of dysarthria.

Mutism may be a symptom of mental disease, and no diagnosis of aphasia should be made until one is certain that it is not a case of mental derangement. The mental reduction spoken of by Marie as the basis of all aphasia is by no means a systematized derangement of the mind, but rather a slight reduction along certain lines.

TREATMENT. Re-education is the remedy, and often touch is better than the presentation of the object to sight alone. Sometimes the vibrations of the larynx of the instructor, as in teaching the deaf and dumb, are the only possible means of awakening the impaired faculties.

CHAPTER VIII

NEUROSES AND DISEASES WITHOUT ASSIGNABLE LESION

RAYNAUD'S DISEASE.

LOCAL ASPHYXIA, SYMMETRICAL GANGRENE.

DEFINITION. This is an affection of the peripheral vascular system, which manifests itself in a spasmodic and recurrent contraction of the arterioles. While it is a general condition, it is most evident in the extremities. It may occur independently, or as a feature of other diseases, such as hysteria, tabes dorsalis, syringomyelia, tumors of the spinal cord and spinal roots, epilepsy, exophthalmic goitre, scleroderma, neurasthenia, myelitis, and insanity, especially with acute mania.

SYMPTOMS OF A TYPICAL CASE. A poorly nourished, or neurotic woman, by or before the thirtieth year, begins to have excruciating pains in the arms, or in the lower limbs. A numbness comes in two or three fingers or toes. They become bloodless and shrunken, they are anesthetic, and if pricked do not show the injury by hemorrhage. After this condition has recurred a number of times during a few months, the parts become gangrenous, and finally undergo mummification. The condition is apt to be symmetrical, and if the feet have at first been affected the hands are attacked later to a somewhat similar degree. The disease may stop at any point, or recede at any stage.

DIFFERENTIAL DIAGNOSIS. This must be from Chlorosis, Frost-bite, Peripheral Neuritis, Senile Gangrene, Gangrene from Embolism, Phlebitic Gangrene, Diabetic Gangrene, Ergotism, Erythromelalgia, Leprosy, and Ainhum.

AGE. Most often between twenty and forty-five, but has been seen in nursing infants, and in old men.

SEX. The cases are females in the proportion of three to one, but those occurring in old age are generally masculine.

ETIOLOGY. All cases present evidence of a neuropathic constitution, and on this basis emotional or physical strain, prolonged lactation, anemia, suppressed menstruation, exposure to cold, or trauma, seem to have acted as exciting causes. A few cases have been attributed to morphine, or chloral intoxication. It has

been known to follow the infections, and syphilis, and is very often associated with scleroderma. The most common causative condition has been a congenitally small aorta.

PATHOLOGY. The arteries and veins have shown no changes which indicate that primary changes in them are the cause of the disease, and it has therefore been ascribed to some perversion of the nervous supply to the vessels. It has therefore been localized by some in the sympathetic system, as a result of a primary disease of the spinal cord, which left the sympathetic system without adequate control.

SYMPTOMATOLOGY. Raynaud divided the symptoms into three stages, viz.: local syncope, local asphyxia, and local death. Normally the first symptom is bloodlessness of two or three of the fingers, and less often of the toes, preceded by hyperesthesia, which is followed by anesthesia, and an absence of hemorrhage from the prick of a sharp instrument. This condition may last from a few minutes up to several hours, and may be associated with chilliness, but no fever in any stage, nausea, and a feeling of general illness. This stage may not develop into the second, but may pass away, only to recur the next day, or in a few hours. After this stage the parts experience a reaction, and become red and puffed, instead of shrunken and cold, and with a stinging pain. After this the normal appearance is regained. Regression may occur at this stage. Several days may intervene between the attacks, but the interval may be marked by an intense burning pain which often precedes the initial attack. After several repetitions of the initial stage known as "Dead Fingers," or this initial attack may develop into it, the affected digits, after a cyanotic stage of an hour or two, become blue black. This is the second stage of local syncope, or regional cyanosis, which may be present in one extremity, while another is the first stage of simple coldness with a waxy-white color. The more common evolution of the disease is to find the toes upon both feet in relatively the same condition, and after they have suffered a loss of the terminal phalanges by mummification, to have the process manifest itself for the first time in the fingers, or vice versa. After the second stage has been reached several times, and been followed by regression into the period of puffy redness, we find that the condition is progressive, and that small blebs and bullae may form, and slight hemorrhages may occur upon the pads at the extremities of the affected digits. These blebs are elevated, and ulceration follows, and then the parts become the seat of a dry gangrene. A line of demarcation forms, and the slough is thrown off in a few months. Suppuration is always slight. This does not necessarily involve a great amount of tissue, and commonly the patient emerges from the disease with the loss of the terminal phalanges on one or two toes or fingers, but sometimes it will recur, until, as in a case of the author's,

successive amputations have been carried as high as the knee. This degree of severity in one extremity does not imply that the other members will be as severely, or as extensively involved. Bilateral attack and symmetry are almost the certain rule in the extremities, but while in addition to the extremities it may attack the inner aspect of the calves, the malleoli, the buttocks, the abdomen, the cheeks, and the tip of the nose, or ears, or may cause lividity of the breasts, it does not, in those localities, often produce gangrene. The genitals and the tongue are rarely attacked. As the process goes on to the more advanced stages the pain becomes constant and excessive. I have known a patient to keep the feet protruded from a window in the coldest weather to seek relief from the distress. The general symptoms are depression and gastric disturbances in the prodromal stage. During the attack, sensation may be lowered in the affected parts, and movements become clumsy, on account of swelling in the soft parts, and rigidity in the tissues, muscles, and skin, and the patient may not be able to walk. Trophic disturbances may be discovered in skin, nails, muscles, and even in the bones. The superficial temperature of the affected parts sinks far below that of the rest of the body. Impairment of sight, hearing, and tinnitus have been noticed. Owing to the spasm of the coronary arteries, collapse sometimes occurs, and since the arteries in the kidneys are subject to the same influence, we find occasionally albuminuria, and glycosuria; hemoglobinuria has also been seen. Oculo-pupillary derangements, and paralysis of the iris, are sometimes present, owing to the action of the sympathetic, but aphasia is rare.

COURSE AND PROGNOSIS. These attacks may be slight, and recurrent through many years, whenever there has been an emotional strain, or a gastric derangement. The ordinary case lasts four months, and terminates in a mutilation of far less extent than the gangrenous area would indicate. A whole foot may become gangrenous, and yet the actual loss may be confined to the two terminal phalanges of one or two toes. In infants the gangrene may come on so rapidly, and be so extensive as to cause death, and in the aged a like danger lurks in the probability of a resulting uremia.

DIAGNOSIS. The diagnosis rests upon the presence of local asphyxia, of all degrees up to gangrene, which has developed slowly with many recessions, and is symmetrical. There has been a neuropathic constitution, and the definite condition has been excited by some mental or physical strain. It is explained by no primary disease of the heart or blood vessels, and the presence of hemoglobinuria, or uremia, will add to the probability that it is Raynaud's Disease. The relative youth of the patient, and the peculiarity of the sensory disturbances are characteristic.

DIFFERENTIAL DIAGNOSIS. Chlorosis is a general, and not a

local manifestation, and it is marked by the absence of sensory disturbances, and particularly by the absence of any intense degree of pain.

Frost-bite is similar in all its immediate manifestations, but the history is too definite to allow an error to be made.

Peripheral Neuritis is symmetrical, and has sensory manifestations of all grades, but they are not paroxysmal, and the nerve-trunks are sensitive. The pains and paresthesias are developed along the areas of distribution of definite nerves, while those of Raynaud's Disease are diffuse. Peripheral Neuritis never eventuates in gangrene.

Senile Gangrene is a disease of old age, and is dependent upon recognizable changes in the vascular system. It is not paroxysmal, and while Raynaud's Disease confines its final destruction to a few phalanges, senile gangrene involves the loss of a whole toe, and in many cases the whole foot. In case that Raynaud's Disease does involve a greater area, it almost always does so by initial attack, while senile gangrene tends to spread insidiously to any extent. There are rare exceptions to this general statement in the case of both diseases.

Gangrene from Embolism or Phlebitis is not paroxysmal, but has a definite localization from the first. These cases are not symmetrical, and have an ascertainable origin in valvular lesions, or cardiac thrombi in the embolic cases, while phlebitis leads to a moist form of gangrene.

Diabetic Gangrene has a history of glycosuria. Failing that knowledge, the case presents inflammatory symptoms, and a rapid, and not a paroxysmal course, or it has followed a traumatism, or there have been notable trophic disturbances of a nervous origin.

Ergotism. This has similar symptoms, but in cases of poisoning by ergot the gastro-intestinal disturbances have been of a very marked character.

Erythromelalgia gives rise to similar symptoms in the first two stages, but not often to the final one of gangrene, and it is commonly relieved by cold applications, which are not palliative of the pains of Raynaud's Disease.

Leprosy. The specific alterations of leprosy are lacking, and in this disease several fingers are attacked simultaneously, and fall off without pain.

Ainhum affects the toes more often than the fingers, is practically painless, and the slough is not preceded by blebs, or bullae.

TREATMENT. No very definite plan of treatment has ever been developed, except upon very broad lines, all agreeing that the person should be well fed, and carefully guarded from cold. This avoidance of chill is particularly to be emphasized in respect to the parts affected, and these should be kept warmly wrapped. If

it is within the means of the patient a warm climate should be selected. While food should be made the subject of special attention, it has been pointed out by one author that the use of stimulants is of doubtful expediency in the case of a neurotic person, since they so easily develop a reliance upon them. Lukewarm baths to the parts have many advocates, and the subject of massage is treated with a large divergence of opinion. Some say mild massage of the parts, others as strongly affirm that it is harmful, but that general massage of the neighboring parts is beneficial. It is said that one of the diagnostic points between this disease and erythromelalgia is that that is relieved by cold applications, while Raynaud's Disease is made worse by them. A patient of my own, however, experienced considerable relief by keeping the feet out of the open window in very cold weather, and afterward seemed to somewhat abbreviate the run of the disease by immersing the extremities alternately in very hot and very cold water, for one minute in each. If the parts are exceedingly painful a 50% solution of Menthol in alcohol may be applied, cotton wrapped over this, and a final covering of oiled silk. Another method of controlling the intense pain is by applying a tourniquet to the limbs, or an Esmarch's bandage. When this is taken off the parts fill with blood, and the vascular spasm subsides. Still another method of producing relief from pain is by applying the positive galvanic electrode to the parts with the negative upon the spine. The affected part may be placed in a bowl of water, in which the positive has also been placed. The static spray has also been directed upon the affected parts, for the relief of pain. With the idea that either the spinal cord directly, or through its influence upon the sympathetic system, has been the cause of the disease, spinal galvanization has met with much favor, with the negative stationary upon the sacrum, while the positive is moved up and down the whole length of the spine, with a current strength of about fifteen milliamperes, for a period of not more than five minutes, care being taken that the current is not interrupted. General static treatment also seems to produce good results in some cases. The line of demarcation should be awaited before operation, as much more tissue may be saved than appears probable from the amount of apparent gangrene.

THERAPEUTICS. *Secale cornutum* in a somewhat low potency is probably the best remedy that we have to offer for the disease. The peculiarity in its symptomatology is the fact that it is one of the remedies affecting the tip of the nose. In the course of the process toward the development of gangrene marked by the production of blebs and vesicles *Arsenicum*, *Lachesis*, and *Crotalus* should be useful. *Glonoin* has been advised, but it must have been on absolutely physiological, and not homeopathic grounds. There is a report by Halbert of a cure by *Ferrum phos. 6x*.

PHYSIOLOGICAL REMEDIES. These have generally been confined to measures to relieve pain, and the use of tonics. In addition to its tonic value, quinine has been advised if the patient falls under the suspicion of previous malarial infection. Nitrite of amyl, and nitroglycerin have been employed to overcome the arterial spasm, and thyroid extract is extolled by Short.

ERYTHROMELALGIA

DEFINITION. This is a vasomotor neurosis of a persistent type, which manifests itself by a transient flushing of the extremities, swelling, and attacks of burning pain, or extreme tenderness, all being very greatly aggravated by a dependent position, and by heat.

THE SYMPTOMS OF A TYPICAL ATTACK are these: A male of early adult or middle age, engaged in exhausting work, and liable to exposure to extreme heat or cold, after an illness which has debilitated him, finds that the feet become reddened if allowed to hang down for any great length of time, or if he stands for a long time. Swelling ensues, and an exquisite sensitiveness follows, or attacks of pain. This occurs only on provocation at first, but soon is excited with inadequate reason, and it may become constant. At an early stage of the disease, rest for a few days perhaps benefits him for a long time. The feet are usually first attacked, but the hands are frequently implicated at a later date.

DIFFERENTIAL DIAGNOSIS. To be distinguished from Chilblains, Raynaud's Disease, and Vascular Spasm.

AGE. Usually young adults, or persons in middle life, but children are not immune (a teething child was the sufferer in one case), and the writer has seen one case in advanced life.

SEX. Females are in the great minority.

ETIOLOGY. It has so often occurred in persons whose occupations were exhausting, and exposed to great variations of temperature, that this has been considered a cause. It has followed severe blows upon the foot; prolonged use of a member when the person is in an anemic condition; and the disease may ensue years after the exposure which put a long-continued strain upon the vascular system in those parts. All toxic and auto-toxic states may predispose to it, as well as anemia, malaria, and syphilis. It has been associated with, or followed the debility from general diseases, the puerperium, and degenerations of the nervous system.

PATHOLOGY. Its pathology is undefined, since by equally good observers it has been ascribed to changes in the posterior parts of the cord, by others to those in the anterior. Others again detect its origin in changes in the peripheral nerves, while still others trace its symptoms to changes in the intima of the arteries. On account of the fact that all these changes have been found in some cases, and are absent in others, doubt of its existence as a disease entity has also found favor. It is probable that at least two classes of cases exist; the first is where the vasomotor irritability results from a primary disease of the spinal cord, and the symptoms are most marked in the extremities, since there nutrition of the nerve-fibres is at its lowest point. The result of this vasomotor irritability is the production of changes in the intima of the arteries of the extremities. The second class of cases rests upon a series of primary changes in the vasomotor nerves at this point, and these changes are sometimes so slight that even the microscope will not reveal them. Weir Mitchell points out that the pure type of the disease can only be expected in young people, since in older persons there may be co-existent arterial and nervous changes which do not have their origin in this disease, but which contribute to its symptomatology when it has been inaugurated. From some of the cases reported it seems probable that exhaustion of the vasomotor centers may sometimes be followed by a peripheral degeneration.

SYMPTOMATOLOGY. We find in all cases, in varying proportion, pain, redness, and swelling. The pain is usually the earliest symptom to be noticed, and its most frequent sites are the great toe, the heel, the ball of the foot on its inner or outer side, or the metacarpo-phalangeal articulation. At first this pain is only apparent after an erect position has been maintained for a long while, or the limbs have been hanging down, or there has been a physical exhaustion, or the weather has been very hot, but after the disease has progressed for a period, the necessity for an exciting cause becomes less and less evident, and the pain becomes more constant, but there is always some part of the day when the condition becomes very much more tolerable, and when the pain may entirely disappear. At any stage of the disease elevation of the limb, and the application of cold will relieve the pain to some degree. The character of this pain is typically a burning one, of almost any grade of intensity, and lacks the piercing and tearing quality which we ascribe to affections of the nerves. Deep pressure increases it more than superficial, and pressure on the nerves may excite it. The redness begins to be evident a few weeks after the appearance of pain, and is of all grades and varieties, according to the period of the crisis. At first it is a flushed look, rosy red, which streaks up the limb in some cases, but is often quite sharply demarked at the ankle. As the crisis deepens in intensity, the

flushing changes to a darker red, like that of intense congestion, and still later may assume the bluish violet of a cyanosis. This persists for a time after the subsidence of the pain. In some cases, for a time between the attacks, this is replaced by an ashy whiteness, with a wrinkling of the skin, indicative of total local asphyxia, produced by the perfection of the arterial spasm. The swelling is of gradual onset, occupies the parts which are the seat of the pain and redness, and is so increased by motion that any use of the affected parts is often impossible. It is peculiar in this, that it generally does not pit on pressure. When the swelling takes place, there is an elevation of the temperature of the surface of two or three degrees. This group of symptoms may last a few hours, or days, or weeks, and then there will be a period of relief of greater or less length. The seat of attack is not often symmetrical at first, but is often so at a later period, and while the disease tends to attack the lower extremities, it may involve the hands primarily, or as a point of secondary seizure. The redness, swelling, and pain are sometimes seen higher up the limb, but usually, whether in the hands or feet, they do not exist above the ankle. Other accessory symptoms are of more occasional occurrence, like the exudation of innumerable drops of perspiration upon the skin at the time of the most acute pain. Hyperesthesias and paresthesias are of uncommon occurrence, nor are weakness and paresis of the parts affected essential to the disease. Trophic lesions are of frequent occurrence, and are of many different kinds; the skin may be thickened, pigmented, and edematous, and nodules and papules on the reddened areas are common; the joints and nails are subject to trophic changes. Ulcers, and fissures, or rhagades may also develop on the extremities. The process may go on to gangrene. Starr denies these trophic changes. Electrical response may be quantitatively diminished.

COURSE AND PROGNOSIS. The local changes are to be expected in only about 20% of the cases. The disease is essentially chronic, and very gradual in its development. It may last for many years, and be strictly limited to one part of one member. It may spontaneously recover.

DIAGNOSIS. This is based upon the presence of burning pain, later redness, and then swelling of one or all of the extremities, when these symptoms are not accompanied by a rise in general temperature, or by pitting of the swelling, and when this whole symptom-complex displays marked intermittency in its progress. It is considered a diagnostic point of great value that at almost any stage the condition is relieved by the elevation of the part, and by the application of cold, immersion in icewater being a very efficient method of its application.

DIFFERENTIAL DIAGNOSIS. Chilblains are similar in appearance, but are marked by a much lower grade of severity, and by

no tendency to increase the length or the frequency of the cycle, and they are present in cold weather, and absent in hot weather, at which time erythromelalgia may be most aggravated.

Raynaud's Disease is distinguished by the fact that while the pain (generally absent, and if present, comes and goes without reference to the position of the part), the redness, and swelling may be present in Raynaud's Disease, they are symmetrical, the pain is relieved, and not aggravated by warmth, or position, and it is very certain to be followed by gangrene.

Vascular Spasm, sometimes termed "Dead Fingers," is to be differentiated by the difference of the location of the condition, the pain is only a dull one, and not burning, there is no swelling, and the redness, if present at all, is only a preliminary stage, but there is a blanched appearance, which condition is a persistent one throughout the attack.

TREATMENT. This is generally summed up in the elevation of the limb, and the application of cold water. Amputation of the part has sometimes been followed by complete relief, as it did not tend to extension, or localization in another part. In cases following injury of a definite nerve, exsection of a part of the nerve was followed by a cure, but in cases from general causes it has not resulted in cure, and in one case was followed by gangrene of the site of operation. Good feeding may prove curative.

THERAPEUTICS. There is no record of cures by remedies to a further extent than some degree of alleviation of the symptoms. Graphites and Petroleum seem to be the best indicated remedies, and in regard to Graphites it has an additional appositeness from the fact that it has cured painful fissuring of the skin when it has occurred from other causes.

EPILEPSY.

IDIOPATHIC EPILEPSY, FALLING SICKNESS, MORBUS SACER.

DEFINITION. Definition is difficult, since the disease may express itself in major, minor, and psychical types. A typical Major or Grand Mal attack is a sudden brief recurrent discharge of nervous energy, not due to a normal cause for such discharge. It results in a brief tonic, and later clonic spasm of a generalized type, accompanied by unconsciousness, and followed by signs of physical and mental exhaustion.

THE SYMPTOMS OF A TYPICAL CASE. The person will at intervals be prostrated by a convulsion which will begin in a tonic form, and end in a clonic spasm. It will begin in a part, and

finally involve the whole body in the spasm. During this convulsion, foam will be ejected from the mouth, the sphincters will be relaxed, the tongue will be bitten, and consciousness will be lost. The attack will last from one to three minutes, when the person will fall into a deep sleep, from which he will awaken somewhat confused, and mentally and physically exhausted.

DIFFERENTIAL DIAGNOSIS. To be differentiated from Focal or Jacksonian Epilepsy, Intoxications, Hysteria, Migraine.

RACE. It is confined to no climate or latitude, or racial division of the earth's population.

SEX. Taking the cases as a whole, no distinction can be drawn. In the first 10 years males predominate, in the second 10 years there are more women. In 913 cases 59.2% were male. (Seibold).

AGE. It may attack the child in infancy, and has been known to occur at the seventy-fifth year, but after the age of thirty it usually results from traumatism, alcoholism, or syphilis. In 913 cases 83% were attacked before the twentieth year. (Seibold).

HEREDITY. Direct heredity is more common than in any other cerebral disorder. Echeverria, out of 533 cases, found direct inheritance in 29.72%; Gowers found it in 35%; Spratling in 66%; Seibold out of 913 cases 34.8%. Those with heredity take it earlier than those without. (Seibold.)

ETIOLOGY. Directly from an ancestor, or indirectly from a defective nervous endowment. Whatever influence may act as an exciting cause, it always pre-supposes nervous instability in the subject. Intra-uterine influences, both inflammations and intoxications, are powerful causes, and if an infant should develop convulsions during the first six weeks of life, they should be attributed to pre-natal influences upon the developing brain; but after that, they are either reflex or toxic. If a child from five to seven years of age develops epilepsy, syphilis should be suspected.

Alcoholism in one or both parents has been considered the cause in about 10% of the cases. (18.8%, Seibold.) He also says that alcoholism in parents is many times more potent than is the indulgence of the individual. Spratling considers that epilepsy developing before the tenth year is probably from bad heredity, cerebral palsy, or is the sequel of an infectious fever. In the period from the twelfth to the sixteenth year inclusive, it is the result of the changes of puberty; this is the particular age for the developement of essential idiopathic epilepsy, but is not so efficient as an exciting cause in the case of males as in females. Fright has been considered to be a sufficient cause in about 5% of the cases. If we presuppose a hereditary weakness in the cortical cells from alcoholism, insanity, or chronic intoxication in an ancestor, any long-lasting peripheral irritation may develop an idiopathic epilepsy. If a person at any stage of life suffers an injury to the skull which will either compress or irritate the cor-

tex, a focal epilepsy is likely to result. (10%, Seibold). After a variable period of life, depending upon the neuropathic tendency of the patient, this may produce a generalized epilepsy in no particular differing from the epilepsy arising from other causes.

PATHOLOGY. The discovery of a center in the pons, the irritation of which would produce general tonic convulsions, and the fact that irritation of the motor cortex will produce clonic convulsions, has led to divergent theories concerning the localization of the lesions necessary to produce epilepsy. Autopsies have revealed many cases where there were changes in the cornu Ammonis, also in the medulla; cases in which there has been an overgrowth of neuroglia in various parts of the cortex have stimulated the belief that the lesion of this disease had a local habitation and a place. Examining, however, the results of 3000 autopsies upon such cases, it was found that while a large percentage of the brains were undersized, or asymmetrical, or showed lesions in the localities mentioned above, there was still no such unanimity between convulsive seizures and any definite lesion as would be necessary to justly consider the one as being caused by the other. Opinion has generally crystallized into the belief that it is due to some general cortical condition, and this belief is strengthened by the fact, that if an epileptic suffered from a cerebral hemorrhage, which injured the internal capsule of one side, the paralyzed side of the body was no longer a participant in the former general convulsions. Since an attack of epilepsy has often been seen dependent upon some prior fault of digestion, or indeed seeming to primarily arise from gluttony, it has seemed possible that it might be the result of an auto-intoxication acting upon a neuropathic constitution. The mechanics of the cortical theory is that there is a degeneration of the second layer of cells, inhibitory, in the cortex; that the motor cells of the third layer, lacking the former inhibitory restraint, respond after a period of rest to peripheral stimuli in irregular and disproportionate motor spasms, until the period of exhaustion supervenes. Be this as it may, it is a well-known truth that there is a periodicity of convulsions in a given case, and, on the other hand, that symptoms of indigestion are apt to precede the motor outbreaks.

SYMPTOMATOLOGY. After a study of 800 patients, Onuf says that there is no absolutely characteristic syndrome, but there is a sufficient conformity to a common type to make it useful in diagnosis. The attack may be preceded for some hours or days by irritability, or, on the other hand, by apathy, either mental or physical, but while this is possible, it is not the rule. Much more common is a premonitory symptom of a definite kind for each person, and usually a persistent one throughout the duration of the case. This is termed an aura, and may be a disturbance of general or special sensation, or be psychological. It is very commonly

a sensation of vertigo, or a bad feeling in the epigastrium rising to the throat, or a sensation in an extremity, usually with the same ascending tendency. These physical sensations may be replaced by voices, or sounds, or visions, and it is common that whenever any of these phenomena have reached a certain point, as a warm sensation from the stomach reaching the head, that the attack of convulsions will be inaugurated. The face now becomes pale, the eyes are thrown upward, the body becomes rigid, with the arms held away from the trunk, the forearm flexed on the upper arm, the fingers strongly contracted, but the legs extended. This stage lasts about twenty seconds, and is the stage of Tonic convulsions, and is usually preceded by a low tremulous groan, occasionally by a run of two or three steps. In any particular case, this tonic period is apt to be marked by a peculiar movement, or group of movements, peculiar to itself. At this stage the patient has fallen, or is just about to fall without regard to his personal danger or comfort. Now comes the stage of Clonic convulsions, which consists in violent and jerking movements of the face and body, of a varying degree of violence, but often sufficient to injure the person severely from contact with surrounding objects. Cases have been seen where this was so violent as to throw a patient from the bed and dislocate a joint, usually the shoulder. During this period the pale face has become deep red, or cyanotic, and this congestion may be so violent that there are hemorrhages in the conjunctiva, and the capillaries of the cheeks. The tongue shares in this jactitation, and the jaws are opened and snapped together with great force, from which we get the familiar symptom of the biting of the tongue, the scars upon which are often diagnostic of previous attacks. This rhythmical spasm affects the throat also, and so we find ejection of saliva, often stained with blood from the bitten tongue. In some cases there is relaxation of the sphincters, not altogether dependent upon the violence of the attack, but sometimes present, and sometimes absent. The pupils are sometimes dilated, sometimes contracted, and differ in the same way in their reaction to light and accommodation. Consciousness is not absolutely abolished, and irritation of the conjunctiva will bring a person again to consciousness, only to relapse so soon as the irritation ceases. Immediately at the attack the knee-jerk is abolished, it then is exalted for a very few moments, and afterward is absent for several days. Ankle-clonus may be present for a time, but in a large number of cases great variation of the reflexes will be found. The plantar-reflex is almost always absent for a time after the attack. The spasm is not usually as well marked upon one side of the body as upon the other. The whole period of tonic and clonic spasms combined is usually only a minute or two, and this is generally followed by a deep sleep, lasting from a half an hour to two or three hours, from

which the patient awakens exhausted in body and mind, but usually mentally clear, and very often hungry, and ignorant of what has occurred. Many attacks are nocturnal, particularly in the early stages of the disease, and they may persist for some years, occurring at frequent intervals, without any suspicion being entertained of their true nature. In the inception of the disease there is a considerable degree of variation in the frequency of the attacks. When they arise from pre-natal injuries, or marked hereditary taint, and become manifested in the first few years of life, they are apt to be repeated at intervals of a week to a month or two, for a year or thereabouts; they then disappear until they recur at puberty, or in late adolescence. They then take the type which is common to cases originating at that age, and at first occur only three or four times a year, but grow more and more frequent until they establish their own peculiar periodicity. Spratling says that out of 1374 cases of all grades of severity, the following frequency was established: weekly or oftener, 57%; every two weeks, 13%; every three weeks, 4%; every four weeks, 18%; every six weeks, 2%; every eight weeks, 3%; every twelve weeks, 4%. The large increase at the monthly interval corresponds to the expected result of menstrual irritation.

Minor or Petit Mal attacks are found alone, or in combination with the major or Grand Mal seizures. These consist in attacks of momentary unconsciousness, forced movements of various kinds, either of the whole body, or of some motor function, or some vocal ejaculation. They are liable to be very frequent, perhaps many in a single day. If found alone, they prognose the later appearance of major attacks.

Psychic Attacks, or, as they are sometimes called, Epileptic Equivalents. These are certain psychical conditions which, to some extent, take the place of epileptic convulsions. They may be momentary, like the motor attacks, or they may last weeks and months, and take the form of bizarre actions, or breaches of manners, or morals, the commission of a crime, or the undertaking of long journeys, and the complete change of one's means of livelihood. They may be inaugurated, or terminated, by a convulsion, by an injury, or illness, or be entered upon, or ended, without any outward influence or manifestation. A certain man may leave his work, and invade the privacy of utter strangers; he may start from his home with a cry, and run through the fields in a great circuit; or change his habitation, and entire mode of life. A patient of the author has several times deserted, to its ruin, a very profitable business. It is a sufficient reason to judge that such unusual actions are epileptic equivalents, if in the family history there have been similar occurrences, interchangeable in the same, or other persons with forms of genuine epilepsy, or if these outbreaks display definite epileptic characteristics, such

as suddenness of onset, transitory duration, and an irregular periodicity.

Status Epilepticus. Sometimes one convulsion does not terminate the attack, but they recur at short intervals for days. This is termed the "Status Epilepticus," and often proves fatal.

PROGNOSIS. It is not a self-limited disease, and opinion has been inclined toward considering it almost incurable. This has been the result of the impression made upon literature by those who believe in epilepsy as an entity, and in bromides as the only treatment. The latest investigations have emphasized the fact, that it is not epilepsy, but one of the epilepsies that we are considering, and consequently that the management, hygiene and medication must vary with the type of the disease. Turner says that the discovery of the bromides has not bettered the prognosis, and Spratling says that he has yet to learn of a single case of epilepsy cured by the bromides alone. At present we may consider, that, even in chronic cases, five per cent. of cures may be expected, and taking cases together, both recent and chronic, the percentage of cure is held to be from 10 to 25%. Sex has nothing to do with this probability, nor has the history of inheritance; but epilepsy beginning in infancy or childhood, or when its approach is delayed until adult life, has an unfavorable prognosis. The epilepsy of old age is quite amenable to treatment, while that beginning at puberty responds most readily of all. The earlier a case is brought under treatment, and the longer the interval between attacks, by so much is the probability of cure increased. Major attacks are more amenable to drugs than minor, and nocturnal than those occurring during the day. Sometimes a remission which promises cure may be terminated by an accident, childbirth, or an inflammatory disease. If improvement is to result from treatment, it is usually manifested within a year from its inauguration. The mental impairment which so often results, may, with some probability, be attributed to the effect of the immoderate use of drugs; however that may be, mental impairment is more likely to follow minor attacks than the major, but a combination of the two most surely provokes it. Females are most likely to develop epileptic dementia. A few authorities stoutly deny the optimism expressed in this section.

DIAGNOSIS. If it were demanded that a picture should be framed to which all cases should conform, diagnosis would be impossible, since there is no one symptom which is absolutely pathognomonic of epilepsy, but upon the positive side a definite statement can be made with few chances of error. The case is one of idiopathic epilepsy if a person, after uttering a sharp cry or groan, runs a few steps, or grasps some near-by person or object, and then, with a pale face, has a tonic convulsion for twenty or thirty seconds, and then falls, not slides, to the ground. He then,

for a minute or so, is thrown into violent clonic convulsions, during which the face is congested, the tongue is bitten, bloody froth is extruded from the mouth, the urine, and perhaps the feces, are voided unconsciously, and the clonic stage is terminated by convulsive grunting. All this time the patient is unconscious, and after the attack falls into a deep sleep. The tonic, preceding the clonic stage is diagnostic, if we see the patient early enough to observe it; the fact that the patient bites the tongue, and in the fall and subsequent convulsion frequently injures himself is strong corroborative evidence. Other points will be better developed in the differential diagnosis.

DIFFERENTIAL DIAGNOSIS. Organic Brain Disease may produce convulsions of a general character, but they are more severe, tend to last much longer than a minute or two, which is the invariable rule of epilepsy, and they recur in groups far more frequently than is true of epilepsy.

Focal or Jacksonian Epilepsy differs from the Idiopathic form principally in the fact that the former has the clonic preceding, or alternating with the tonic form of convulsion. The focal convulsion is also longer in duration, and is less apt to be general in its distribution. A focal convulsion rests upon the presence of a definite material source of irritation of a definite area of the cortex, the history of whose inception may be discovered, or the evidences of whose existence may be perceived. The convulsions are often as frequent as one or more daily, during the first year of their establishment, while such a frequency is never seen in the idiopathic variety.

Intoxications may develop generalized convulsions with quite a similarity of symptomatology, but here again the frequency is greater, the sequence of symptoms is not so regular for the given case, and while the epileptic is quite apt, especially in the early stages, to be in robust health, the sufferer from an intoxication sufficient to produce convulsions is the subject of a cachexia.

Hysteria is such a protean disease that there may be a close resemblance. Points of difference will be found on examination. In the convulsion the movements are wider and more purposeful, and yet, withal, the patient does not expose himself to injury, either when he falls, or when he throws his limbs about in the convulsion. The attack is not marked by the same degree of unconsciousness, nor is it so uniformly terminated by a deep sleep. The tongue is not bitten, although the lips may be; the eyes are not fixed, but are in constant motion, and the lids flutter; the kneejerks are exaggerated, while the plantar reflex is lost, and the sphincters are not relaxed. The termination of the attack is often marked by a profuse passage of colorless urine. On examination of the urine, it will be found that in hysteria the urea, phosphates, and ethereal sulphates are reduced greatly, and albumin may

appear. It is not uncommon for an epileptic attack to be followed by an hysterical seizure.

Apoplexy may be suggested to one when the patient is found in the comatose condition, but epilepsy is a disease of youth, with no signs of arterial degeneration; the face by this time has lost its congested appearance; the coma is not so deep but that the patient can be aroused; the pupils are generally even; there is no sign of paralysis when aroused. Positively, we find that the tongue, head, or limbs of the epileptic show scars from former attacks, and hemorrhages on the conjunctiva and the cheeks are common. A delay of an hour or two terminates the coma of an epileptic, while it still persists in the case of the apoplectic. A history of former attacks is generally obtainable in the epileptic.

Migraine may exist with epilepsy, or alternate with it. Migraine may have an *aurea*, but its duration may be a quarter of an hour; that of epilepsy only a few seconds. The most frequent one in migraine is a numbness or tingling in the arm, followed by hemiparesis of the opposite side, and if it is of the right arm, aphasia may follow both affections. The *aurea* of migraine is never in the foot. The most frequent form is some visual disturbance. Lateral hemianopsia is frequent in migraine, but is never seen in epilepsy. The pain of migraine may be followed as in an epileptic attack by sleep, and therefore might be confusing.

TREATMENT. The modern treatment of epilepsy does not consist in the administration of any one drug, but in putting the organism into the best condition for resisting the tendency of any peripheral irritation or internal intoxication to cause a violent and unregulated explosion of nervous force. Individualization of the case is the keynote of modern treatment. It has been already said that the continuous action of a peripheral irritant might be transmitted in some way to the brain, and produce such a weakened condition of the cortical cells that generalized convulsions might be the result. This is not only theory, but clinical experience, and therefore every cause of such irritation should be removed. The removal of spurs from the septum, adenoids, defects in the muscle-balance of the eye, growths and cicatrices in the rectum, urethra, uterine cervix, and about the clitoris have resulted in actual cures in some cases. Spratling says that he has never seen the correction of visual defects by glasses produce any lasting effect, and Jacobi makes the same statement concerning circumcision. While few will claim that any one product of metabolism, like cholin, is the cause of the disease, all agree that perfect digestion and excretion are at the root of any possible cure, and at times seem efficient for great mitigation of the attacks. Vegetarianism has few advocates, but all agree in limiting the use of meat. Dechlorinization (the absolute elimination from the diet of sodium chloride, and the substitution of bromide of sodium in cookery even) has been

strongly advocated, but it cannot be persisted in for more than four weeks without grave danger to health, and especially to mentality. It has been decided that its actual benefit is the increase in the efficiency of the bromides. The reduction of salt to a reasonable minimum is endorsed by all. The person should live an open-air life, with an enjoyable occupation, which is as free as possible from probability of danger if the patient is overtaken by an attack. The diet should be plain, avoiding indigestible foods, the tough skins of fruits, or fruits themselves if of very firm texture. Eating should be in moderation, and food taken very slowly, since many epileptics are inordinate eaters. If poorly nourished, use cod-liver oil, but never alcohol. The rooms inhabited and slept in should be cool, since the contrary condition has been known to precipitate an attack. Plenty of water in the diet. Since epileptics notoriously suffer from lowered vitality, the extremities should be kept warm, and the body fully protected.

Electrical Treatment. Uniform opinion is that whatever success has been achieved depends upon cephalic galvanization; the only difference of opinion on this point is as to the method of application of the galvanic current to the head. Erb also recommends general faradization, but lays the greatest stress upon cephalic galvanization. Some of the successes have been obtained by passing the galvanic current through the motor centers of the brain in the neighborhood surrounding the fissure of Rolando. Others by passing the current transversely through the temples, while still others have passed it in every conceivable direction. Erb advises placing the cathode on the nape of the neck, and the anode on the side of the forehead, passing a weak current for about one minute, and then bringing the anode to the forehead, and cathode to the occiput, giving the same current in strength for the same length of time. Althouse and others recommend galvanizing the seat of the aura, which as a rule is in the abdomen, and may easily be brought under the influence of the cathode in central galvanization, while the effect of the anode is exclusively on the head. (King.)

THERAPEUTICS. *Agaricus.* Where the attacks are characterized by the exertion of great strength.

Artemesia absinth. Is efficient in attacks of petit mal, where consciousness is not entirely lost, where attacks are repeated many times in a day, and where there is giddiness with a tendency toward falling backward, and a persisting tremor.

Argentum met. Seizures like epilepsy followed by delirious rage, in which he jumps about and tries to strike those about him. The probability is that the cases reported have been hysterical.

Argentum nit. It has helped some cases, and is particularly applicable to those occurring at the menstrual period, or from fright. The attack is preceded by dilatation of the pupils for days

or hours before the attack. The attack is followed by restlessness instead of sleep, and there is general tremor.

Arsenicum alb. This is advised in such cases as have, preceding the convulsions, a burning sensation in the stomach, pressure and heat in the back extending to the nape of the neck and brain, with dizziness. The stupor following the convulsions is broken by restless agitation.

Amyl nit. Has been recommended for use by olefaction, when the convulsions are preceded by marked flushing of the surface.

Borax. This remedy has been used without very precise indications, but is of value when a patient presents a general nervous impressionability. Nervous instability is its keynote.

Bufo rana. This is especially indicated where the attacks are in the young, are marked by great periodicity, and result from the debilitating effects of masturbation. The attacks are ushered in by a cry, and the face is livid. The attacks are preceded by a marked mental irritability, and the patient may be paraphasic. The aura is in the solar plexus. Spasms better by warmth.

Calcarea carb. Epilepsy about puberty. Prescribed principally upon constitutional symptoms.

Causticum. Nocturnal attacks, especially petit mal. Involuntary urination, in the stage of stupor. Great faintness and weakness following the attack.

Cina. Nocturnal epilepsy with opisthotonos.

Cicuta vir. Convulsions are nocturnal, frequent, easily recurrent from the slightest touch, and followed by prostration similar to that found in Causticum. Great faintness and weakness following the attack. The aura is replaced by a venous congestion, photophobia, and mental confusion, and the pulse is slow. The countenance is markedly cadaverous, and the jaws tend to become locked.

Cocculus. Convulsive attacks preceded by the characteristic symptom of hollowness or emptiness of the whole body, the head especially.

Cuprum met. The attack is preceded by signs of mental irritation, anger and irascibility; then follow the manifestations of a similar motor state; the fingers and toes twitch, and there is a chewing motion of the jaws. The attack itself is simply a development of this motor irritability carried to the extent of general rigidity, and perhaps opisthotonos.

Digitalin. In doses of one grain of the 3x every other day has been recommended for attacks arising from excessive nocturnal emissions.

Hydrocyanic ac. For attacks markedly tetanic in character. It has been termed by some a specific.

Hyoscyamus nig. Epileptic attacks preceded by the characteristic mental condition. There are twitching of the jaws, and a

chewing motion, and during the attack a bluish countenance, and involuntary urination.

Ignatia. Violent convulsions with an alternately red and pale face. Suitable for young women, and those who are of an excitable temperament.

Indigo. Where epilepsy is known to arise from worms, and the patient is of a sad and anxious disposition.

Kali mur. This was Schüssler's standard remedy, especially for cases arising after the suppression of an eczema, or other eruption.

Lachesis. When the attack is preceded by cold feet, eructations, paleness of the face, vertigo with heaviness of the head, and palpitation of the heart. The patient screams out, and the limbs jerk, and the fists are clenched. Clinical results have been good in some cases.

Lycopodium. Epileptic attacks of the usual type, but with extreme periodical anxiety.

Melilotus alba. This was highly recommended by Doctor Bowen, but has not found very general endorsement. The indication is an extremely severe type of headache preceding the attack.

Nitric ac. Nocturnal attacks preceded by a sensation as though a mouse were moving up and down the left side.

Nux vomica. Such cases as seem to arise directly from indigestion.

Opium. This has been recommended for nocturnal attacks, with violent convulsive movements of the limbs, and symptoms of suffocation.

Plumbum met. The characteristic symptoms are a paralytic heaviness of the limbs before the attack, and, after a prolonged sleep period, a temporary motor paralysis of the lower limbs. A cure is reported of a case which always appeared at the menstrual period, with colic, obstinate constipation, and a sallowness of the complexion.

Secale cornutum. Is highly recommended in rapidly recurring paroxysms, with rapid sinking of the vital powers, and paralysis of voluntary motion.

PHYSIOLOGICAL TREATMENT. Moon says urethane and chloral have been the best remedies.

Dr. J. M. Bennion says that Potassium bromide is the drug most commonly employed; the sodium salt is more soluble, less irritating, and less depressing to the heart. The Strontium salt has the same characteristics, and is still more efficient. Thirty grains in an ounce of water, thrice daily, control attacks better than the mixed bromides of potassium and soda. It does not produce a rash, acts best on female patients, does not injure the mental condition, in fact seems to improve it. All this relates to the epileptic *nisane*.

Kellner used baths and opium with bromides with apparent advantage. For fifty days patient is given three times a day .05 of gramme of extract of opium. This dose is increased by .01 g. every other day, so that by the 50th day the highest dose of .29 g. is reached. On the 51st day 0.3 g. is given, and this concludes the opium treatment. Bromines are now given. Noon and evening of the 51st day 2 g. are given of a bromide mixture, one part bromide of potassium, one of sodium, and a half part of ammonium; on the 52d and 53d days 6 g. of this mixture are given; on the next two 7 g.; on the next two 8 g.; from the 58th day on, 9 g. are taken daily for a long period of time. During the opium treatment the patient takes three times a day a tablespoonful of 1% hydrochloric acid three times daily, and Carlsbad salt as needed. During the opium period a bath is taken daily; the first day, temperature 24° C. and lasts 10 minutes; on the second day, 23° C. and lasts 9 minutes; on the third day, 22° C. and lasts 8 minutes, and so on until the eighth day the temperature is 17° C. and lasts 3 minutes. This is continued until the 15th day, and from the 16th to the 23d, the bath lasts 4 minutes; 24th to 31st, 5 minutes; 32d to 50th lasts 6 minutes. In addition, the usual hygienic treatment of fresh air, diet, and employment. He subjects to this treatment only those who can stand the opium dosage, and whose mentality is well enough preserved to make a return to the normal possible.

Alfred Gordon believes in the gradual withdrawal of starch. Nitrogenous food and stimulants are absolutely forbidden, the quantity of starchy food is reduced to a minimum; milk, vegetables, water, eggs, fruit and bread are the only articles allowed, and bromides given. Without bromides its effects are short-lived. It rests upon the fact that elimination is more perfect. He was led to believe that the toxic retention was the cause of the attack, from the fact that when methylene blue was given a few hours before a seizure the urine was clear during the attack, and only attained the bluish-green color after the attack.

Lundborg, of Upsala, said that the dechloridization seemed to affect many cases favorably, but not altogether without a bad effect upon them.

Salonggi, Damo, and Zambelli advise a salt-free diet, or rather reduced salt, and say that it stops the lighter attacks, modifies the severe ones, and with the addition of bromide may stop even them.

Arthur Morton, after a use of several years, says that the substitution of sodium bromide for sodium chloride does not affect nutrition; it does constipate, but with intelligent patients is useful, in those with poor control useless, in the demented is advantageous.

Schnitzer reduces salt to 30 grains in 24 hours; bread should

contain instead of salt sufficient sodium bromide to give the patient 90 gr. a day in 13 ounces of bread. In addition 3 pints of milk, $1\frac{1}{2}$ oz. of butter, 3 eggs without salt. The patient will endure it for about six weeks, and often gains in weight. Jules Voisin and Roger Voisin, and M. L. Krantz say that dechloridization has no lasting effect upon the number of seizures. It does intensify the action of the bromides. By the 4th month patients broke down, became the prey of various hallucinations of sight, taste, general sensation, and the delusion that they were being poisoned. They also had dyspepsia, great fatigue, disinclination to work, lumbar pains and muscular cramps.

Hugues believes in silver nitrate on account of its power of intestinal antiseptics. Believes that perfect digestion is the most promising method of treatment.

Peterson believes that an indiscriminate use of the bromides is worse than the disease, and that when used it should be in small doses. Combine with some of the harmless remedial agents, or instead of the bromine salts some preparation of brominized oil, which is less powerful, and less noxious.

Spratling says that he has yet to see the first case of epilepsy cured by the bromides alone; at the Craig colony the average dose is 15 grains a day, 5 grains to the dose. He quotes a case which had taken for years 120 grains a day to 140 with no effect, but with improved hygienic care he made a complete recovery on 15 grains. It is never necessary to bring on a bromic acne.

D. R. Brower insists on intestinal elimination to perfection; for antiseptics, salol with phytolacca; for cerebral irritability, sodium bromide, never over 60 grs. daily, in plenty of water. Sometimes adds $\frac{1}{2}$ to 2 dr. of *Solanum Carolinense*. Strychnine valuable for meeting circulatory and vasomotor disturbances, and also commends fluid extract of *Adonis vernalis*. Cerebral sclerosis calls for alteratives.

J. Hermann Branth believes in the use of the Röntgen rays on the skull.

S. La Place believes in appendicostomy, so as to flush the colon.

Rinne had a case where the seizures always began with twitchings of the right thumb and hand. The patient was trephined, area of the cortex over that area 3 or 4 mm. thick, and 1 by 1.5 mm. in area was excised. Since then attacks have been very much lightened, and have become more infrequent. Is not certain whether the result came from relief of compression, or from excision of the cortex.

Friedreich reports on 11 cases, 1 cure, 2 very greatly improved; benefit in some of the others from trephining, and removal of a portion of the dura mater. Thinks it proper in cases known to have a traumatic origin, and in others where it is known that cortical irritation exists.

St. Clair Thomson reports removal of adenoids in a child of six, and relief of epileptic convulsions.

Jules and Roger Voisin assert that no particular variety of food is harmful, but that complete digestion and some bromides are needed.

M. Delagénère tied off the superior longitudinal sinus one-third of an inch behind the fissure of Rolando, and cured a case of epilepsy. This was done to repair an injury during a surgical operation. He afterwards performed this operation with premeditation, with good results.

Epilepsy of malarial origin can be cured by the use of 6 grains of quinine daily for several days.

Doctor Audinino and Doctor Bonilli, of Lombroso's clinic, Turin, after using various preparations of calcium by mouth, and hypodermatically, chiefly calcium bromide, have thought it was superior to potassium bromide and other methods of bromide treatment, and attribute the value of milk diet to the amount of calcium brought into the system.

R. O. Moon says that borax is often valuable where potassium bromide has failed. This is denied by many experimental tests.

Branth, as stated by Manders, recommends X-ray treatment, three treatments a week; 5 minutes exposure, 15 inches distance, decreased to 10 inch distance for 10 minutes. A hard or high vacuum tube, backing up a stop-gap of 5 to 8 inches. The alopecia is only temporary. May be best to continue bromide with it. Not advisable if degeneration of the brain has begun. Treatment is followed by gain in weight, improvement in faulty enunciation, less severe, and less frequent seizures.

Turner. Epileptics suffer notoriously from lowered vitality, and sluggish circulation in the extremities, for which warm baths, spinal douches, and massage are important remedial agents.

CHOREA.

ST. VITUS DANCE, CHOREA ANGLORUM, CHOREA VULGARIS, SYDENHAM'S CHOREA.

DEFINITION. It is a spasmodic affection of the nervous system, limited in duration, and characterized by jerky incoördination of voluntary movements, involuntary twitchings, and some muscular and mental weakness, varying largely in different cases. There are several varieties, Huntingdon's Chorea being a hereditary form, seen in several individuals of a family, accompanied by nervous degeneration, and usually ending in insanity. The Electric chorea of Dubini, known only in Italy, appears to arise from a specific micro-organism, and is rapidly fatal. Habit-choreas, or

tics, are involuntary spasms of isolated muscles, and never organize into general disease.

THE SYMPTOMS OF A TYPICAL CASE. A person in childhood, or early youth becomes physically weak, develops irregular jerking movements in a member, or successively in all parts of the body, which may involve the tongue, face, and muscles of deglutition, and phonation.

DIFFERENTIAL DIAGNOSIS. To be differentiated from Metallic Intoxications, Toxic Poisons, Hysteria, Multiple Sclerosis, Friedrich's Ataxia, Athetosis, Post-hemiplegic Chorea, Huntington's Chorea, Tics, Myoclonus.

RACE. Is almost exclusively Caucasian, and cases have been said to be especially frequent in the upper classes, but the somewhat wide experience of the author indicates that privation and faulty hygiene cause as large a number of cases among the poor.

SEX. Two or three females to one male.

AGE. Rare before four years, or after twenty. An enormous preponderance before the fifteenth year.

ETIOLOGY. Causes are predisposing and exciting. The predisposing causes are practically stated when we say that a person has a neuropathic heredity. Some say that it is caused by anything that lowers the nervous vitality of a neurotic individual, since, in consequence, a toxin may be evolved, which has a selective action upon the cells of the motor-cortex. The rheumatic diathesis, acting in conjunction with the especial vulnerability of a neurotic and degenerative heredity, is the cause most commonly seen. Many doubt the assumed relation to rheumatism, since in a large proportion of cases it cannot be detected.

One writer has traced chorea in one family for five generations. The chief point of interest is in the fact, that in the fourth generation, in which the chorea reached its most intense stage, one child had reached the age of fifty years without betraying any signs of the disease, and he alone was not nursed by the mother, since she died soon after his birth. He asks: Cannot the deficient nutritive character of the milk of a choreic mother have a detrimental effect upon the child, and cannot the tendency be weakened by other and better nutrition? The other five children of this mother all developed chorea under the age of thirty years. The deduction from many recent sources is this: that the exciting cause is some unknown toxin, which is capable of producing both the choreic, and joint, and cardiac symptoms. A strong argument against rheumatism being the exciting agent is that an actual rheumatic history is placed by some as low as 18%, and also that the joint symptoms are fully as liable to follow, as to precede the choreic symptoms. In one anemic patient who was in the eighth month of pregnancy, the urine was found to be loaded with indican. Under better feeding she recovered, and went through her preg-

nancy successfully. In a case of a girl of twenty-three, pregnant, with syphilitic secondaries, anti-syphilitic treatment stopped the attacks, and she completed her pregnancy successfully. It is therefore evident that the exciting causes may be quite numerous and diverse, but a micro-organism similar to that of rheumatism is the cause of a large proportion of the cases.

PATHOLOGY. No uniform changes have been found, nor has any constant micro-organism been isolated. The site of the lesions discovered has been in the cortex, rather than in the cord. In the brain there have been found intense hyperemia, perivascular exudation, minute hemorrhages, occasional emboli, and spots of softening which tend to be unilateral. Doubt is cast upon the existence of specific Chorea-bodies, and also upon the theory of Pick and Kahler, which is that choreiform movements are caused by irritation of the pyramidal tracts. If this were true, it would be difficult to explain the frequency of Hemi-chorea. All evidence being given its due weight, the deduction must be drawn that the cortical cells are constitutionally deformed, and that in the presence of any form of debility they fall into a state of irritable weakness, and the muscular twitchings are the result.

SYMPTOMATOLOGY. There is usually a prodromal period of malaise, insomnia, restlessness, peevishness, poor appetite, constipation, and mental dullness; this latter is the most common symptom, but the emotions may be too readily excited. Next is to be seen an inability to preserve a steady contraction of the muscles. It may be only a quivering of the eyelids, or a lack of dexterity of the hands, but usually in all cases there is an unsteadiness upon the feet. Movements may be voluntarily suppressed for a time in mild cases. Most marked of all, but often unobserved, is the readiness with which fatigue is induced. We may find bone or joint pains, and the premonitory malaise continues. The muscular movements resemble voluntary motions, but they are purposeless, and are jerky, and begin usually in the hands, thence go to the forearm, and perhaps affect the face. The disease may begin on one side, and go over to the other. The method of the protrusion of the tongue is diagnostic, in that it is snaky, and the lips being also affected, speech may be very much affected. The decrease of muscular power is very marked, and there is a form called Limp Chorea which is almost paralytic. The sphincters are never involved, except in the last stages of fatal cases. Electric excitability is often increased, and may show a moderate reaction of degeneration. Mental Fatigue is easily produced, and we may get capricious emotional expression, and also stupidity. In a grave type we are apt to find hallucinations and delirium of some grade, and this is quite the rule in adult cases, and those complicating pregnancy. Sometimes cases go on to paranoia. Two cases of Dementia choreica have been reported. These cases

had the walk of paralysis agitans, and the spasms were of a major type, with minor intermixed. There was no rhythm, and they acted like jumping-jacks. One of these cases was traumatic. The mental symptoms were Irritability, Seclusiveness, history of Persecutory ideas, characteristic speech defects and progressively increasing dementia. The general agreement now is, that the more chronic forms, such as Huntingdon's, have many points in common with hysteria and epilepsy, and have a kindred pathology in the cortex. Reflexes are normal, but may be jerky. If tactile sensibility is much blunted the case is probably hysterical. In the heart we find some form of disorder in 65% of the cases. These disorders range from irregularities of rhythm to valvular lesions, usually mitral. The alteration of rhythm is usually ascribed to cell degeneration in the nuclei of the pneumogastric.

COURSE. An attack usually lasts about four weeks, and one third of the cases show recurrence, to which females are more liable. These recurrences are usually three in number, each one being shorter, and the interval between being longer than the preceding one. The last remaining symptom is a twitching of the face, or protrusion of the tongue.

PROGNOSIS. Recovery is the rule in cases occurring before the tenth year, but there is a fatality in plain uncomplicated cases of 21-2%. When it thus occurs, autopsy sometimes fails to reveal an adequate cause. Complicated cases die from exhaustion from excessive muscular effort, inability to take food, endocarditis, and other heart complications, cerebral hemorrhage, concurrent affections. The chorea of pregnancy is fatal in 25% of the cases.

VARIATIONS ON THE ORDINARY TYPE. Chorea Gravis, or muscular insanity which is more fatal, since the violence of the spasms exhausts the patient, and prevents the taking of nourishment.

Choreic Status where, like Status Epilepticus, the spasm continues for days, and the patient may die of exhaustion.

Limp Chorea where paralysis is the most marked symptom; monoparesis is the most common form, the muscles are lax, reflexes are abolished, but atrophy is rare.

DIAGNOSIS. Depends upon the developement of irregular, involuntary, and incoördinate movements of a purposeful nature, even while at rest, beginning in the face or an extremity, and extending over a considerable portion of the voluntary muscles; diagnostically affecting the tongue with a jerking, darting motion on attempts at protrusion, and not being preceded, or accompanied by evidences of an organic brain or nervous disease.

DIFFERENTIAL DIAGNOSIS. Intoxications from poisonous metals or toxins may appear like it, but the muscular fault in such cases is a tremor, rather than a jactitation, and there are associated symptoms from the primary disease.

Hysteria gives rise to rhythmical, not jerking motions, and there are usually stigmata, or a history of antecedent seizures; the patient is apt to be older, and there is a history of a psychological causation.

Multiple sclerosis has similar movements, but they are volitional. While the eyes twitch in chorea there is no defined nystagmus.

Friedreich's Ataxia. This has an unsteadiness on the legs and in gait, which simulates the gait and station of chorea, but the peculiarity of incoördination does not appear while the patient is seated, and at rest. The deep reflexes are apt to be diminished, and the disease is familial.

Athetosis. The movements are slow and even, and not jerky, although they do appear while the patient is at rest. There is a history of some antecedent causative disease.

Post-hemiplegic Chorea. This has the history, and the diagnostic marks, of a preceding hemiplegia or diplegia. There are spasticity, and increase in the deep reflexes.

Huntingdon's Chorea. This generally has a family history, and is accompanied by some degree of mental deterioration. In the familial form it occurs after middle life, and the other form is of the senile period.

Tics or Habit-Spasms. These are of single muscle-groups, and show no tendency to extension.

Myoclonus is usually a disease of adults. It is also usually located, or begins, in the lower extremities, and is bilateral, the face being spared as a rule. The muscular movements are very rapid, reaching the rate of 60 to 100 per minute, and a tonic stage may supervene.

TREATMENT. Rest in bed is the best of all measures, as exhaustion is such a pronounced factor; this is peculiarly true of cases from the lower walks of life, where it is often due to improper hygiene, and lack of proper food. Isolation is also necessary in severe cases, and forced feeding, and massage, and warm baths at 100 degrees to promote sleep through as many hours of the twenty-four as is possible. After movements have ceased, the rest and careful hygienic treatment should be continued for an additional two weeks. Medicines are of subordinate importance. On the other hand accurate prescribing may obviate the necessity of any especial hygienic care, other than the avoidance of exhaustion, and is especially called for in cases where hygienic conditions have been good. It is not so certain as the first method, and severe cases will demand the skillful exercise of all the means at our command. In those severe cases called Muscular Insanity, where the patients throw themselves about continually, and with great violence, it is necessary to pad all the projecting parts of the bed, and all the joints of the patient. Even this will not always

obviate abrasions and bruises, and in such cases I have sometimes found that the best results will be obtained by placing the patient upon a couple of mattresses spread upon the floor, where the amplitude of the space seems to have a good effect upon the violence of the movements, and also takes away some possible sources of injury.

Electrical Treatment. Electricity has strong advocates, and it is claimed that the presence of great muscular weakness indicates its value in all cases, and that the earlier it is begun the better. The only question, therefore, is the method of its application. In cases of Muscular Insanity, it is advised that the anode be placed over the cortex, and the cathode in the hand of the affected side, using a mild current for three minutes. If the jactitation is universal, repeat the galvanic application on the other side. Since the nerve cells which are specifically weakened are those of the motor cortex, Erb advises that general faradism should be employed by placing one pole over the fissure of Rolando, and the other pole upon the neck upon the opposite side of the body. Riggs claims that a persistent facial chorea may be benefitted by a faradic treatment, gradually increased to such a strength as to slightly fatigue the muscles. Galvanism is employed as follows by Riggs: He passes a current longitudinally through the head, giving two to three milliamperes for two or three minutes, and then along the spine for seven or eight minutes with a strength of ten milliamperes. Cohn recommends local galvanization of the affected parts with currents of three milliamperes, the cathode being placed in the jugular fossa, and an anode of smaller area being moved over the affected parts. Galvanization of the sympathetic in the neck is also efficacious. The anode is placed below the lobule of the ear, posterior to the ramus of the jaw, the cathode is placed in the jugular fossa, and a current of one to three milliamperes is employed for a few moments. The cathode may be placed upon the opposite side at the jugular fossa, or in the region of the transverse processes of the lower cervical vertebrae. The static current is sometimes employed alone, or in combination with galvanism. Inasmuch as most of the patients are of tender years, it is not advisable to employ sparks until the patients have lost their fear of the treatment. King advises that it be first given by negative insulation only, allowing the charge to pass off from the hair, and other points, the charge must be a light one, and sparking must be avoided, or the choreic movements will be increased. At later treatments a breeze along the spine may be employed. Certain habitual choreic movements, which remain after the general twitching has passed away, may be removed in a week or two by a static spray over the spine. Riggs says that excellent results occur in chorea from first giving central galvanism followed by static. During the first treatment there may

be a slight apparent aggravation due to nervousness and fear of the treatment, but with care in the administration this will soon disappear. King states that that form of choreic movement which occurs in adults, and is persistent, and does not respond to general treatment, is not benefitted by any form of electricity. This is naturally true, since it is not a true chorea, but is a result of organic changes from cerebral palsy in childhood.

THERAPEUTICS. Aconite. Cases from emotional causes and characterized by great fear. It has been recommended in alternation with Gelsemium.

Actea racemosa. It is good for cases displaying symptoms of trouble in the joints, and for patients about the age of puberty.

Agaricus. This is very often the remedy. A very neurotic disposition is an indication for its use. Agaracin, 2x., a one-grain powder every second hour has been curative.

Arsenicum. For idiopathic cases. The greatest use has been in tangible doses of Fowler's Solution.

Argentum nit. This has been advised, but the grounds are doubtful, since it is marked by tremor rather than twitching.

Asafetida. For very delicate and nervous children.

Asarum. For cases where the whole body, and limbs especially, feel light, and where the cutaneous nerves are super-sensitive.

Belladonna. For neurotics who are very excitable, with marked vasomotor symptoms. It is used in potency, and also in doses of from one to five drops of the tincture every four hours.

Causticum. For cases arising from fright, and also those which would seem to demand Ignatia, but are not relieved by it.

Cina. If the condition proceeded from worms.

Calcarea carb. This is usually given upon constitutional grounds, but O'Connor states that he cured a very severe case with the 200th.

Cocculus. This is advised, but is a spinal, and not a cortical, remedy.

Cicuta. This evidently acts upon the spinal cord and medulla, and is not applicable.

China. When anemia is the evident basis of the malady.

Cuprum met. and ars. For idiopathic cases. It is the first choice of many observers.

Crocus. Excessively neurotic patients.

Dulcamara. When the disease has been precipitated by exposure to wet.

Ferrum. Where it is apparently a vasomotor neurosis. When used for anemic characteristics, give Ferrum redactum, one grain, three times a day.

Gelsemium. Given for very severe cases, either alone, or in alternation with Aconite. One author says that in doses of one to five drops every four hours, it is as efficacious as Fowler's Solution.

Hyoscyamus. In very severe motor cases. Such patients are very weak, with a tottering gait, and seem to have an erroneous impression of distances, reaching for distant objects as if they were near.

Ignatia. A remedy given on general grounds, for cases with an hysterical element.

Iodine. If there is a tuberculous strain.

Mygale lassiodora. This is for the light and semi-hysterical cases. Very useful in such.

Nux vomica. Much used, but probably aids by the general tonic power, as does *Strychnia*.

Phosphorus. On general indications.

Phos. acid. Hartmann quotes the cure of a case of great severity.

Pulsatilla. Simultaneous affection of the joints.

Rhus tox. Under the same conditions. Its value is over when they are improved.

Santonin. Where tapeworms, or intestinal worms, are the cause.

Sepia. In children of weak and flabby fibre who take cold easily, and are emotional and despondent.

Spigelia. This may do good where the joints are affected.

Stramonium. Good in the very severe convulsive types. It may be followed by *Cuprum* in the later stages. In one of the author's cases this remedy was curative of chorea gravis, after Arsenic liberally administered by a previous attendant had been a total failure; given in tincture, 10 drops in a half glass of water, teaspoonful hourly.

Sulphur. As an intercurrent.

Tarantula hisp. For hysterical types.

Veratrum vir. In plethoric girls.

Zinc. A feeling of great illness, and mental depression.

Zinc phosph. Valuable in the 3x, for nocturnal attacks. May be useful in material doses.

Zizia. Useful if the cerebral circulation seems greatly at fault.

PHYSIOLOGICAL THERAPEUTISTS have settled upon three drugs to the exclusion of all others. They are Arsenic, Ergot, and Asparin. In relation to the first two it is said that some cases are benefitted by one, some by the other, and still others do best by a combination of the two. Taylor and Spiller both doubt the harmlessness of Arsenic, and both have seen neuritis and idiosyncrasy follow its use.

Arsenic is almost always given in the form of Fowler's Solution, and is begun with an initial dose of from 3 to 5 drops, 3 times a day. It is increased a drop a day, until toleration is exceeded. This is shown by slight nausea, or pain in the stomach, or puffing under the eyes. It is never best to push it to the extent

that vomiting occurs. When the symptoms of intolerance occur, one or other of two courses is pursued. One method is to wait until these symptoms subside, and then begin again with the original dose, while the other is to begin with the dose which was being administered when the toxic symptoms first manifested themselves. Of these the first plan is the better. F. M. Pope says: Certain cases should not be treated with Arsenic. He divides them into three classes:

1st. Those who are obviously unsuited. These are: Very acute cases with coma or paralysis; those who have been treated for some time with small doses of Arsenic; those who have an active rheumatic process; cases showing advanced cardiac disease.

2d. Cases which are intolerant of treatment.

3d. Cases which resist it, or who are unbenefitted by it.

In all other cases, Arsenic should be given according to the following rules:

Tongue must be clean before beginning the treatment, and if not, some mild mercurial purge should be given.

Put the patient upon a bland and digestible diet.

Give the drug well diluted, and diluted in the same way throughout.

Do not stop at the first attack of vomiting, since it may have been accidental.

Increase the dose daily.

Keep the patient in bed during the treatment.

If the vomiting persists, stop the drug for 24 hours, and then start with the same dose.

Examine the patient daily for toxemia.

Riviere uses the following with constant success in all except those cases of the most violent type, in which only control can be exerted: Fl. Ext. Ergot, 1 drachm; Liquor Arsenicalis, 3 drops. This is one dose. Increase this as it may be needed. He bases his therapy on the division of choreas into three classes:

1st. Those benefitted by Ergot.

2d. Those susceptible to Arsenic.

3d. Those where one drug seems to be necessarily supplemented by the other.

Asparin. Children bear it very well, and 10 grains may be given to a child of thirteen, every four hours. It should always be given in an acid medium, and never in an alkali, never in hot fluids, but preferably dry or in lemon-water, but may be given in cold milk. This is better than Arsenic.

PARALYSIS AGITANS.

DEFINITION. This is a disease which has been classed among the neuroses, on account of the lack of any constant pathological

findings. It is characterized by a general rigidity of the muscles, with a great deal of apparent weakness. This rigidity distorts the body and the limbs in flexion, and produces the characteristic attitude, and gait. The face loses its expression, and since mental operations are performed as slowly as are the physical movements, the patient appears to have some degree of dementia. The extremities, and perhaps the whole body, are affected by a fine and rapid tremor.

THE SYMPTOMS OF A TYPICAL CASE. There is an increasing rigidity of the whole body, without an increase in the deep reflexes, a tremor which is fine and constant, except in sleep, and is passive in type; the gait is trotting, the face is expressionless, and the speech is high-pitched and monotonous.

DIFFERENTIAL DIAGNOSIS. It is to be distinguished from Multiple Sclerosis, Simple Tremor, Senile Tremor, General Paresis, Hemiplegic Tremor, Toxic Tremor, Traumatic Neurasthenia, Senile Paraplegia.

AGE. It is a disease of the latter part of life, and the vast majority of cases occur between the age of forty to sixty years. This rule is however not an invariable one, since well-known observers have reported cases as young as twelve, and a large number, in the aggregate, before the twentieth year.

HEREDITY. It is not classed among the hereditary diseases, but while direct heredity is not so common (Bury reports two cases, Borgherini three, Gowers two, Pennato two, and others, here and there, in literature) there is always evidence of a neuropathic condition in the parents.

ETIOLOGY. This depends upon a predisposing neuropathic condition, which may be inherited, or may have been acquired. While it has often been noted that it is a disease which seems to pass the profligate by, and attack the industrious and virtuous, equal stress has not been laid upon the fact that those industrious and virtuous persons have, almost uniformly, so lived and worked as to reduce their vitality, and render the tissues extremely liable to degeneration. The exciting cause may be a sudden or a long-continued strain upon the emotions, or the muscles; a definite injury to nervous tissues, by traumatism, or the action of infectious diseases. Fright and anxiety have both been followed by it; it has followed prolonged night-watching, where the loss of sleep was made more detrimental by co-existent anxiety. Occupations, involving the prolonged use of a limited number of muscles, are a frequent exciting cause. A prolonged, unusual strain upon a single set of muscles (carrying a heavy bag some miles was the cause of one case), has often been followed by the disease. Krafft-Ebing found that out of one hundred and ten cases, he could find seven where the disease had directly followed the injury of a particular nerve, and first appeared in the area of

innervation of this nerve. It has closely succeeded illnesses from infectious causes, particularly after typhoid; somewhat marked as the exciting causes in the unusual adolescent cases.

PATHOLOGY. It has been called a disease without a lesion, but this should be qualified by adding the adjective "definite," since changes have been found in all parts of the nervous system; but they are not constant, nor does their severity, or distribution, give any criterion of the severity, or extent of the disease of which they might be supposed to be the cause. Autopsies have disclosed changes in the muscles similar to those in pseudo-hypertrophy; there is also a tendency to myxedematous overgrowth in the skin of the face, and in patches over the rest of the body; sclerotic changes have been disclosed in every part of the nervous system. On account of the changes in the skin and muscles, some have attributed it to a perversion, or lack, in some glandular secretion, while others, like Gowers, would localize it in the motor-centers of the cortex of the brain. Hughlings-Jackson considers that the disease is a form of hemiplegia, but it is pointed out that the lack of change in the reflexes, and the absence of ankle-clonus, make the position a hard one to understand. Taylor believes it to be based upon a disease of the caudate and lenticular nuclei, and not alone in the cells, but also in the association-tracts. This would ally it with chorea, and make both diseases depend upon an insufficiency of cerebral control over motor discharges. It is thought that the fact that it is sometimes hemiplegic or monoplegic in distribution demands an organic cerebral basis for the malady.

SYMPTOMATOLOGY. The invasion is generally slow, occupying from one to three years in attaining full development. Tremor is the first obvious symptom, and it is initially seen in one hand, and then ascends the arm of that side, and next invades the leg. The same order is followed upon the other side, as a rule, but divergence from this order is not uncommon. Occasionally the invasion is abrupt, but usually it is a system of advance, and recession, and then advance, until the disease is fully localized. Long before any suspicion of the disease is excited, the person feels a weariness and stiffness of the muscles. This is never a paralysis at any stage, since a sharp command will provoke a prompt and adequate response. It arises from changes in the nerve-terminals, and the fibrillae of the muscles, and causes a clumsiness in movement which becomes quite distinctive. It is not a spastic condition, since the reflexes are not exaggerated. This rigidity increases as the years pass, until the whole body is moved as if in one piece. If the patient wishes to change the direction of his vision, he may have to turn the whole body. While the respiratory muscles, and those of deglutition may feel stiff, the functions of both are accurately performed. The muscles

of the face, being affected by the same rigidity, utterly lose their customary play of expression, and if, as is common, there is some muscular hypertrophy, and a thickening of the skin, the mask-like face is distinctive. The muscles of the eyelids, and eyeball, and the iris are all affected by this same weak rigidity, and their motions are clumsy and retarded. The fact that contracture ensues from the changes in the muscle structure, and that this contracture exercises its force toward flexion, produces conditions which are peculiar to the disease. The curvature of the body is forward, the arms drop to the sides, but with the elbows held out from the trunk, the forearms are bent at nearly a right-angle, and crossed over the chest or abdomen; the hands are bent inward on the forearms, and the fingers are bunched, and bent slightly toward the ulnar side. The forefinger and thumb are usually quite closely applied to one another, and this position combined with a nearly continuous fine tremor, produces the diagnostic "pill-rolling" movement. Another effect of this muscular condition is the characteristic gait. We make the ordinary movements in walking for a threefold purpose, viz., to progress in a premeditated direction, to maintain our lateral balance, and to maintain our center of gravity in a fore-and-aft direction. At each minute of our progress we are making numerous delicate and instantaneous re-adjustments. In paralysis agitans the clumsy and weakened muscles fail in all these points, and so the gait becomes a trot, to catch up with the center of gravity, which has been displaced with the first step. Some persons cannot stop, and must run into some fixed object to bring them to a halt. This is called "festination." In some cases, if left alone, they will go indefinitely to one side; this is called "latero-pulsion," and if to the rear, it is called "retropulsion," but this often occurs when they have been walking forward. In arising from a seat they push themselves up by the hands, and in seating themselves fall into, instead of sink into a seat. The contractures referred to as existing in the hands may also exist in the feet, and the flexion of the toes may prove a further embarrassment in locomotion. Tremor is the most characteristic symptom of the disease, and after it appears as a fixed symptom it is constant, never ceasing except in sleep, chloroform narcosis, and shortly before death. In a typical case it appears a few months after the muscular rigidity, but it is the first symptom in cases arising from shocks to the emotions, and is usually in the affected muscles, in those cases attributable to muscular strain, or the injury to a nerve. It is, however, absent in some traumatic cases. It is passive in type, i.e., while the parts are at rest; and disappears on volitional movements of the parts; it is fine in extent, and rapid in rate, i.e., more than six oscillations to the second. In combination, these characteristics are distinctive. While it is passive

as a rule, it is sometimes intention, i.e., increased by voluntary movement of the parts. The rate may be changed, and also the amplitude, by emotion or fatigue. It sometimes is a remittent symptom. It follows about the same rules in invasion and extension as do the weakness and rigidity, but it always spares the abdomen, the head and the jaws. It may be so pronounced as to shake the whole body, giving rise to the idea that the head is also in tremor. Use of the arms in some cases will cause the feet to shake and drum upon the floor. When the head seems to be affected, if the patient is laid on a couch, so that the head and trunk are both supported, it will be found that the apparent tremor of the head was communicated to it by the violence of the shaking of the body. The tongue may show a fine tremor, but the lips are held firmly (tremor has been noted in one case); if the eyelids are closed they may disclose a fine tremor. Writing displays the tremor, as it is wavy, both on the up and down strokes. The muscles of phonation are weakened, and rigid, but do not display a tremor. The weakness and rigidity make the voice high-pitched and toneless, but the "jolting" phonation, sometimes evident, is caused by the general tremor of the body. The sphincters are not weakened, but, in some cases, micturition is hurried. The reflexes are not affected uniformly in all cases, or at different stages of the same case. In the beginning they are exaggerated, as in neurasthenia, and in late stages they are diminished as the rigidity becomes marked, but in no case are they abolished. Foot-clonus is never present. Electrical reactions are normal. The mental condition is unresponsive. It is frequently stated that they are unemotional, and live in a sort of "fool's paradise," but I have always found them easily moved to tears, and, less often, to laughter. They seem to be weak-minded, but this assumption is an error; the inertia, so evident in the muscular system, is equally true of mental processes, and a sudden strong stimulus will, in either case, provoke a normal response. They do not tend to terminate in dementia, but a small proportion of the cases mark the final stage by a mental breakdown, initiated by a rise in the body-temperature. Sensory changes are absent as a rule, but some cases have displayed a diminution in the perception of pain. This loss shaded off into the normal as the trunk was approached. Sometimes we find paresthesias, and, very rarely, a case may be neuralgic. Vasomotor changes are common. These manifest themselves in flushings, sensations of heat and cold, and in palpitation. The patients have attacks of sweating, and this is often localized, yet not always on the parts affected by tremor. The alteration of the secretory functions is also evinced by the increased flow of saliva, and the occurrence of diarrhea.

COURSE. The slow and remitting invasion has been described,

and it may take years for the full picture to be developed. The gradual increase of all the symptoms finally reduces the patient to helplessness, but does not seem to materially shorten the expectation of life.

PROGNOSIS. The probability of cure is almost "nil," but the danger of an abbreviation of life is almost as slight. Even at the late period of life, when the majority are attacked, they have an expectation of life, all the way from ten to forty years. A few cures have been reported, but the author has seen only one case of amelioration even.

DIAGNOSIS. This rests upon the history that the patient had first a weariness, followed by a rigidity of the muscles; that he then developed a fine, rapid, passive tremor, most marked in the extremities, and which spared the head and abdomen. That he had palpitation, flushings, and feelings of heat and cold. It depends upon the fact that the patient is bent and rigid; that he walks bent over and with a trot; that his face and voice are both expressionless; that his pupils are sluggish, and his oculomotor muscles are rigid; that he is mentally unemotional. That with all this, his reflexes are not organically disturbed, and that his electrical reactions are normal. Incomplete cases must be diagnosed on history and symptoms present, taking into account their order of evolution, giving due weight to heredity, and to the age, and occupation, and habits of the patient.

DIFFERENTIAL DIAGNOSIS. Multiple Sclerosis. This disease is most often confused with paralysis agitans because both have tremor, but multiple sclerosis is a disease of those under thirty, while paralysis agitans very rarely affects those under forty. Objectively also; the paralysis agitans patient is contracted in flexure, while multiple sclerosis has no flexure, but has a foot-clonus, staccato-speech, and nystagmus, which are never found in paralysis agitans. The tremor is also different. It is passive and continuous in paralysis agitans, intention only in multiple sclerosis; it affects the head in multiple sclerosis, never in paralysis agitans. Ask the paralysis agitans patient to perform an act, and the tremor fades away as he approaches the goal, while initially the multiple sclerosis patient may display none, but on attempting the desired movement it becomes evident, and grows wilder and more active as he approaches the consummation of his task. In the rare cases where paralysis agitans has a tremor of the intention type, the absence of organically disturbed reflexes, and changes in the optic nerve, will bar the assumption that the disease is multiple sclerosis. The rigidities also differ. Multiple sclerosis has spastic rigidity, i.e., from a sclerosis of the lateral tracts, while that of paralysis agitans is from a change in the muscles (although we do not certainly know how, or whence, it is inaugurated). The first is a spasticity, while the second is a

rigidity. If pressure is exerted upon a spastic limb, it withstands it for a few seconds, and then suddenly gives way, ("lead-pipe" rigidity); put pressure upon a rigid limb and it bends at once, up to a certain point, and never gives way beyond that point.

Simple Tremor. This begins early in life, varies little at the years pass, and markedly affects the head, and often that only, and is often directly hereditary.

Senile Tremor. This mostly affects the head; usually primary there; fine in time and extent. At first it is intention, but later is passive; it does not tend to increase. It is sometimes very difficult to diagnose from the appearance of the tremor, but history may be an aid. The tremor of paralysis agitans begins upon one side, while that of age upon both, simultaneously. Remember that the two conditions may co-exist.

General Paresis. This has a fine tremor, but it differs in several particulars. It is not passive, but evident only when muscles are put into a state of tension. It is then particularly evident in the face and lips. The personal history is different; the reflexes tend to be abolished; the mental state is not apathetic, but is marked by a deep melancholy, or of great exaltation.

Post-hemiplegic Tremor. It has been said that the tremor of paralysis might be hemiplegic, or initially so; this is true of post-hemiplegic tremor. This latter, however, has the history of a preceding apoplexy; if that cannot be obtained, we can find some actual paralysis, there is an exaggeration of the knee-jerk, accompanied by a clonus of the foot.

Toxic Tremors. Sensory symptoms are common in all of these, and, on the other hand, all toxemias may be barred if we find a fixed attitude, immobile face, and festination. Alcohol produces an irregular tremor which is wide in range, and is evident in the legs when the patient attempts to stand. The individual is in a state of constant restlessness. He has foot-drop and gastritis, and his nerve-trunks are very sore.

The tremor of mercury is often the first symptom to appear, and it is very coarse. It is all over, and is intention, but may be constant. It shows first in the face and tongue. The patient has a stomatitis and an irritable stomach. Arsenic shows pain before the tremor appears. While lead has a marked tremor it usually can be detected by a wrist-drop without pain, with much motor-weakness, and the blue line on the gums.

Traumatic Neurasthenia. This may at times produce a similar tremor, but the period of invasion is very much shorter, and, as time goes on, rigidities are hardly ever developed in the neurasthenic case, and the peculiar gait never. The anemic state is similar, but it is amenable to treatment only in the neurasthenic case.

Hysteria. This may have a like tremor, and similar bodily

conditions, but the hysterical patient (except in traumatic cases) is a young female, who has stigmata, particularly restricted visual fields, and contracted, or reversed color-fields. The tremor, while similar, is almost entirely controlled by psychic stimuli. The mental condition is never so placid as in paralysis agitans, and the inception of the case was from a class of excitants which differs from those of paralysis agitans.

Arthritis Deformans. Literature furnishes one case of this disease which presented a tremor of the proper type, and the distinctive attitude, but as this case had also a cervico-dorsal kyphosis the writer questioned whether the tremor might not have been produced by pressure upon the motor cervical roots.

Senile Paraplegia. This depends upon an arterio-sclerosis of the brain and spinal cord. These cases exhibit the posture and tremor of paralysis agitans, but they will also develop some paralyses, and defects in speech, swallowing, urination, etc.

TREATMENT. In some cases improvement has followed the administration of remedies, but the possibility of cure is denied by those whose wide experience, and judicial temper of mind entitles them to speak with authority. Under such conditions we are not justified in urging treatment upon those afflicted with the disease, unless we are convinced that it will stay the disease to some extent, or that it will render them more comfortable in body or mind. Experience has demonstrated that treatment does mitigate many accessory symptoms, the anemia common to all these cases can be benefitted, and the patient is rendered much happier by the knowledge that some attempts are being made to mitigate his condition. Upon these grounds the patients should be urged to live a quiet life, as free as possible from physical strain and worry; he should be as much as possible in the open air. A plain diet should be enjoined, although some alcohol is often a benefit. He should choose an equable climate to reduce the strain of life, although no climate is in any degree curative. The advantages of exercise without fatigue may be obtained from massage, and frequent rubbing with the hand is beneficial. Persons have thought that in the early stages benefit has been derived from suspension in a Sayre's apparatus. Some patients seem better after a carriage ride, attributing this benefit to the jolting. On this account Charcot advised the use of a jolting-chair, or some other method of vibration. Gowers says that this is contrary to common experience. Westpfal advised nerve-stretching, but this has been generally condemned. Lukewarm baths relieve the vasomotor symptoms and reduce the tremor in some cases. These should be combined with electricity and massage. This brings up the question of the value of electricity. It is not considered to be of much curative value, but since many patients will have it, from a charlatan if not from their own

physician, its utility must be considered. King concludes as follows: Electricity is not essential for the purpose of nourishing the muscles, but may be used by passing the stabile galvanic, or even faradic current, through the muscles. An interrupted galvanic current is never indicated. Some results have recently been secured by central galvanism. Little good can be expected in chronic cases, although painful sensations in rigid muscles, insomnia, and anorexia, might be relieved by proper electrical applications. Tremor, and uneasiness of muscles may also be relieved to some extent. Charcot recommends the static breeze with an occasional discharge of sparks, which he states to act particularly well upon the tremor. Laret reports a single case which showed a marked amelioration of the symptoms from the use of the high-frequency current, in connection with the static spark. He also finds that vibratory treatment in connection with static electricity is palliative in many cases. Eulenberg advises a weak current through the head, and Beard and Rockwell galvanization of the sympathetic nerves and the brain. Gray used a large flat electrode to the nape of the neck, and another, somewhat smaller, to the lumbar spine. Current of from three to five m.a. increased later to fifteen m.a. for five minutes, three times a week. It is doubtful if the spinal cord can be influenced by electricity applied in such a manner.

R. Friedlander applies gymnastics and Swedish movements to the relief of the condition. He makes the patient conquer the tremor by directing the attention to the small muscular groups, and later to the larger. As the flexors are most affected, he endeavors to strengthen the extensors by resistive exercises, and so he expects to gradually conquer the rigidity. A natural swing to the arm and leg is next attempted. He makes a slow heel-and-toe-gait, of varying length, an exercise, until the patient can perform it without mental effort. The guide to the amount of exertion is the pulse-rate. Upon any acceleration, exercise must be stopped until it again becomes normal. Several weeks are needed for a sufficient trial of this method.

THERAPEUTICS. We can hope to accomplish very little with remedies and many of the remedies advised in our textbooks, while they have tremor and stiffness of the muscles in their pathogenesis, will be found to be inapplicable, since these will be found to have been spasticities, instead of rigidities. Argentum nit. has clinical evidence of some potency in this disease. Its symptomatology comprises the tremor, muscular weakness and rigidity, flushing and palpitation. Baryta carb. and mur. should be thought of in these cases on account of their efficiency in changes which suggest senility, or pre-senility. Where tremor is the initial symptom we should think of Agaricus or Agaracin in the 2x. Gelsemium has the tremor especially marked, but, at the

best, it can be considered a remedy for the initial stages only, and only then when the vasomotor symptoms are a deficiency, instead of the usual increase in arterial tension. Hyoscyamus has tremor and stiffness of the limbs, and peculiarly the flexor spasm of the toes. It may be of some service. Sparteine 1x, 2x, was given to a patient for the weak condition of the heart, and, unexpectedly, the tremor was greatly improved. Physostigma has been given for cases having a clear sensorium, and a tremor apparently arising from a circulatory disorder of the spine. The following have also been recommended: Arsenicum, Causticum, Ergot, Ignatia, Lycopodium, Mercurius (on the ground that it benefits any nervous degeneration resulting from interstitial overgrowth), Oleum Jecoris Asseli, Phosphoric acid, Plumbum (strongly endorsed as at least palliative of the symptoms), Rhus (when the patient has been rheumatic and the invasion was subsequent to exposure to cold and wet), Stramonium, Scutellaria, Zinc, and the Bromide of Zinc.

Physiological medicine makes use of various tonics, Amyl nitrite (but never Digitalis) for the vascular spasm, and Hyoscyamus, either in the fluid extract, or the alkaloid, as the best remedy for the tremor. Strychnia and some form of Arsenic are considered the ideal tonics, but the Iodides are also largely used. Atropine will help the tremor, but dries the mouth too much.

HYSTERIA.

DEFINITION. This is a disorder of the mind, where there is a contraction of the consciousness, and a weakening of the powers of correct deduction from peripheral stimuli. The conscious mind seems to be weakened, and the subconscious dominates the functions of the body. Hence we find a disordered condition of nutrition, vasomotor and trophic irregularities, and disturbances of the special senses. Moebius has said of it, that it is a condition where ideas control the body, and produce morbid changes in its functions.

THE SYMPTOMS OF A TYPICAL CASE are some form of muscular paralysis, or some general, or special sense-perception, with or without a perversion of the functions of mind or body, or of both. It is accompanied by disturbance of cutaneous sensibility on the trunk, as well as the limbs, and the areas are bounded by right lines. The area of vision is diminished, and there is a reduction in the area, or a change in the order of the relative visibility of colors.

FREQUENCY. It is a common disease, and confined to no one race, but the Celt, the Slav, and the Israelite furnish the great proportion of the cases.

AGE. Most frequent about the age of puberty, and although it has been thought to have been as early as the second or third year of life, it is rare before ten, or after forty.

SEX. Formerly it was thought to be found in the female only, since the name indicated its supposed seat, and cause, viz., uterine; but now it is known to occur in the male, but far less frequently, and most usually in those of the lower classes, and arising from traumatism as a rule.

HEREDITY. Often a direct heredity can be found, but rarely through the male parent. It is more often indirect, in the sense that any debilitating condition in an ancestor predisposes the descendant to hysteria. These ancestral influences may be any of the cachexias, or intoxications, or the practice of vicious habits. Such an influence will of course be intensified, if such taint is common to both parents, and therefore, consanguineous marriages are prolific of such offspring.

ETIOLOGY. The causes are distinctly predisposing, and exciting. Granted that any of the hereditary influences, just stated, have been operative, it is evident that the individual will be hereditarily weak in the nervous system, and will be unable to resist the ordinary shocks of life. Any unusual emotional strain, will be sufficient to inaugurate the hysterical condition. The same cachexias and intoxications which have been spoken of in the case of an ancestor, will operate directly in some cases; masturbation has been judged to be an efficient cause, and secret alcoholism has produced it in adult women. Castration and the climaxis have been followed by it, and such also has been the result of traumatism.

PATHOLOGY. None is exactly known, but it is probable that changes are set up in the cells of the cortex. Whatever may be the final lesions, the process leading to their existence is based upon a nutritional fault. There is an increase of uric acid, xanthin compounds, and urobilin.

SYMPTOMATOLOGY. Generally speaking, there are a disturbance of common sensation, disorders of the special senses, the development of hysterogenetic zones, and, less often, convulsions of an epileptoid character. Some of these symptoms are more permanent than others, or more usually found at all stages, and so they have been placed in two distinct categories. The common, usual and earlier, are called Stigmata, and by other writers referred to as Continuous, while the later group, which are seen later and less frequently, but quite as permanent when once established, are termed Paroxysmal, or Accidents. If either stigmata or accidents appear, they are apt to preserve the same form throughout the disease. The presence of stigmata has been so dwelt upon in descriptions of this disease, that it should be emphasized that all cases do not present stigmata, and that in

their absence the condition must be diagnosed by exclusion, from the fact that the disease simulated by the hysterical person will markedly differ from the actual complaint, either in the combination of symptoms presented, or in the order, or rate of their appearance.

Stigmata may be definite changes in sensation, or power, or control of motion, or of the operations of the mind. The change is generally a loss (anesthesia). It differs from that due to organic disease, in its distribution and outline. It is distributed in patches over the face, trunk and limbs, or of half of the body, or a single limb, or an extremity. In diseases of the peripheral nerves, we may find extremities anesthetic, but the trunk is spared, or until very late in the disease, while in diseases of the cord, the anesthetics will be as they are represented in the segments of the cord, and not scattered here and there, with no relation to such representation; while if from the brain, we shall have an antecedent condition to explain its presence. It will impress the observer, if the area is large, that the anesthesia is of such description as could readily be pictured in the mind, since we can readily imagine an anesthetic hand, or foot, a whole leg or arm, or a half of the body. The outline is occasionally peculiarly irregular, but, in a vast proportion, the anesthetics are bounded by right lines, i.e., forming squares and rectangles, or, if of an extremity, it will have a straight border for its upper limit. This gives rise to the name of "Glove" or "Stocking" form of anesthesia. Such outlines are very diagnostic, when we recollect that the skin is innervated by the expansions of sensory nerves in such a manner that the terminals interlace, so that normal sensation fades into anesthesia with a great indefiniteness in its actual disappearance. These cutaneous losses are possibly to all forms of stimulation, and the patient may fail to experience heat, cold, and pain as completely as he does tactile sensation. In some cases, we shall find one quality of loss in one part, and a different one in another. Another fact is that they may be localized, or shifted by suggestion, or magnets, true or false, or by any other measure that strongly appeals to the mind of the patient. Nor are these losses confined to the superficial structures. There are deep anesthetics affecting the joints and muscles, and the viscera. This gives rise to a sort of hypotonia, and also to a loss of the perception of fatigue. When this is present, the patient cannot state correctly the position of a limb if the eyes are covered, and cannot execute movements correctly, since they are in fact ataxic. The abolition of the perception of fatigue explains the phenomenon known as "*Flexibilitas Cereæ*," a wax-like rigidity, which is seen in the cataleptic state, where the patient will hold a limb in an elevated position for hours, which would be impossible for one in health. The patient is unconscious of these anesthetics, and, as

they are possibly in minute patches, long search may be necessary to establish their existence. The mucous membranes of the mouth, ear, eye, and rectum, and urethra are subject to the same losses. It is diagnostic, however, that it never attacks the irregular area of skin covering the coccyx and its neighborhood, the perineum, and the genitals. Anesthesia of this region is always organic in origin. There is loss of special sense if the organ lies in an anesthetic area.

While loss is the rule, there may be an increased response to peripheral stimulation (hyperesthesia). There may be pain, where simply touch should be felt, or touching one area may give rise to pain in another, but it will be found that the pain upon a light touch is lost, or not increased, when considerable pressure is made upon the part. We may also find cases, which are the subjects of spontaneous pain. Such pains are apt to be localized in parts where injury has been experienced, or is apprehended.

Losses of the functions of the special senses are common stigmata, and sight is the one most likely to be perverted. Contraction of the visual fields is most often seen, but, as it occurs also in neurasthenia, reversal of the color fields (dyschromatopsia) is the most diagnostic sign. A person in health ought to perceive an object brought forward along the side of the head, so soon as it reaches a position at right angles to the axis of the direct vision, i.e., he ought to have a visual arc of 180 degrees, less the loss on the nasal side. The hysterical patient suffers a diminution of this area, all the way to a field just the size of the aperture of the iris (tubular vision). This when seen, is often bilateral, and has been seen in neurasthenia. To test this condition: seat the patient in front of the examiner, and have him fix his eyes directly to the front. Then slowly bring a pencil in vibration from behind the head, toward the front, close to the side of the head, having previously warned the patient to say "now," as soon as he perceives it. By repetition on the other side of the head, a sufficiently clear determination of the field of vision may be obtained. By closing one eye, and moving the pencil forward upon that side of the head, the nasal area of vision, made narrower by the encroachment of the nose, can be mapped out. Do this upon both sides, to avoid over-looking half-vision (hemianopsia), a loss usually from an organic cause. Reversal of the color-field (dyschromatopsia) is discovered by the use of some form of perimeter, and rests upon the fact, that the normal eye perceives white over the whole visual area, blue over a little less extended field, red over still less, while green is the most contracted. In hysteria, this order is often reversed in an eccentric manner, so that, for instance, green may be visible over a larger area than blue. In such a case, however, the revolution of a

Newton's Wheel, bearing red and green, will be seen as white, as in the normal eye, showing that the retina and eyeball are normal, and that the fault is in the brain. Where one half of the body is anesthetic, sight will be lost, or will be very dim upon that side, complete loss being rare, but may come on suddenly, and go as suddenly.

Faults in accommodation may be a symptom, and we may get a diplopia, or covering one eye, the other may see double, or triple objects. Visual losses being present, the conjunctiva, lids, and an area of skin about the eyes will be found to be anesthetic.

Hearing is liable to be perverted to a partial degree, total deafness, however, being almost certainly organic. As in sight, so here, the meatus and skin will be anesthetic. This deafness is not due to a defect in the auditory apparatus, but, as in visual losses, resides in the brain, and is shown to be so by Rinné's test. The normal ear will hear the vibrations of a tuning fork about twice as long, when it is held in front of the ear, as when its heel is pressed against the mastoid process; that is, air conduction is better than bone conduction. A hysterical person gives the same response to the test, showing that the auditory loss is not in the ear, but in the brain. It is said that an hysterically deaf person, in attempts to hear, does not incline the head toward the speaker, as does one actually deaf.

The Voice may be lost, and in such case the larynx is insensitive. The organic, and tendon reflexes, are not modified, as in similar anesthetic conditions from organic causes. The pupils will react to light and accommodation. The iris will dilate upon pinching the skin of the neck, even though that may be anesthetic, and though the sight may be diminished. The abdominal and cremasteric reflexes are normal, and if the skin is pinched where it gives rise to pain, the respiration and action of the heart will be accelerated (Mannkopf's sign). Buzzard says, that the unusual combination of an exaggerated knee-jerk, and a diminished plantar reflex is common and diagnostic. An absolute loss of knee-jerk is never hysterical, but the ankle-clonus may closely simulate that arising from organic disease. Palpebral and pharyngeal reflexes may disappear, if the parts are insensitive, but there is no constant rule, and they may be voluntarily restrained, and tickling the trunk or plantar surface does not produce the usual response. The pharyngeal is absent in 90 per cent. of hysterics.

Motor Stigmata. These should be carefully separated from the phenomena known as motor accidents, and they must be sought for, because the patient is ignorant of their existence, although practically they differ only in degree from the stigmata. Motor stigmata are very variable in their forms of expression, but commonly there is a tardiness in the commonest movements, which rate of response is greatly influenced by the diversion or concentration

of attention upon them. They are also characterized by an incoordination in movement, which is, of course, proportionate to the loss of muscular sense, but is peculiarly independent of the presence of anesthesia, since, with an anesthetic hand, a patient can grasp a pencil even with the eyes bandaged, and indicate its use, although if the anesthesia is complete, there will be a loss in this power. Another feature is the inability to perform simultaneous movements, or those which require divided attention. Voluntary movements are also weaker than is normal, and if a limb has been strongly flexed, or tightly bandaged, or subjected to any treatment which strongly appeals to the mind, it may be the seat of rigidities and contractures, in which case it will be found that the tension is equal upon both flexors and extensors, contrary to the rule in organic complaints. Similar treatment may cure it.

Mental Stigmata. These cases are often characterized by peculiar losses of memory (amnesia), and this loss may relate to certain portions of time, certain groups of facts, or the movements of particular people, and the loss may be so systematized as to lead them into very erroneous statements, and hypnotism will often show these to be purely functional losses. Weakened will power (aboulia) may exist, and it will give a person a hesitancy in performing the commonest acts, and an uncertainty in regard to the fact of their execution. All these people are extremely suggestible, which makes them ready subjects for hypnotism. They simulate every kind of known complaint and condition, not for the pleasure of it, but because they are the prey of an unregulated response to external stimulation, both material and psychic. Contrary to popular opinion, they are not erotic, as most hysterics are sexually cold.

Accidents. These, as has before been said, have some of the characteristics of stigmata, and, when thoroughly established, may have the force of stigmata, but, properly speaking, are sensory, motor, and convulsive attacks of a more temporary and paroxysmal character. Sensory accidents may take the form of a headache resembling that of syphilis or migraine, or that from tumor, or meningitis. There is a hysterical pseudo-meningitis, which resembles the organic disease in the intense pains, the wild cries, and even the opisthotonos. We find all grades of irritability of the spine, and of neuralgia of the viscera, and there is a form of angina pectoris, which is considered to have hysteria as its base, and is not easily diagnosed from the organic disease.

Motor Accidents. The so-called paralyses are practically losses of energy to move a limb, and not loss or impairment of any of the mechanism to perform an action (abasia). Contractures also exist, with or without the presence of pain. One type of this symptom is called Brodie's joints, where contracture is combined with a neuralgia, or hyperesthesia of, generally, the

large joints. It is the most common of all the monosymptomatic attacks of hysteria. A diagnostic point is that the pain is not worse at night. Charcot says that it is diagnostic, that after an anesthetic, muscular rigidity returns first, and pain next. Charcot also says, that in malingering the production of a contracture requires great concentration of attention, strength, and energy, and that, from this exertion, we get great irregularity of movement, and respiration is increased, which symptoms are absent in the contracture of hysteria. The complete monoplegia of a whole member, or of only the hand or foot, with normal movements of the corresponding member, is found only in hysteria, and is either flaccid, or combined with contracture. In such case, the limb will show some contraction of the muscles if the corresponding limb is being used, or in intoxication, anger, or terror, and if the limb is placed in a position requiring voluntary action for its support, it will not immediately fall if support is withdrawn. Rhythmical spasms of limbs, or single muscles, are not uncommon.

Tremor is usually said to be passive, but instances are quoted, where it is as much intention as that of multiple sclerosis. The etiology of the tremor found in hysteria is not always clear, since toxic poisoning, multiple sclerosis, and other diseases in which tremor is a prominent symptom, are not infrequently complicated by hysteria.

Hysterical Convulsive Attacks. Charcot's description is classical. There may be a prodromal stage of mental and physical symptoms, which are apt to be uniform for a given case. The symptoms may be depression, or increased irritability, fear, palpitation of the heart, globus, etc. Then comes what might be termed an aura, rising from the ovaries, from the stomach, or from the throat, combined with marked fear, tinnitus, palpitation, mist before the eyes, and mental confusion. Then we have: (1st) The epileptoid period, where the patient falls, not suddenly as in epilepsy, but still in a tonic condition. (2d) Period of clownism, which is inaugurated by clonic spasm, contortions, and grand movements, opisthotonos, etc. (3d) Passional period, of plastic attitudes, expressing devotion, or passion, or fear, with an expression of face conforming to the movements of the body. (4th) Period of delirium, of a quiet nature, with prominent hallucinations of animal forms. The whole duration is from fifteen to thirty minutes, but the attacks may succeed each other, so that the condition will last for days (the status hystericus). The exhaustion of true epilepsy may bring on an hysterical convulsion. It is to be diagnosed from epilepsy, from the fact that the tonic stage is longer, and opisthotonos is marked; the movements are more active, and betray volition, and consciousness is rarely completely lost. External influences increase the severity of the attacks; the corneal reflex is never lost, the light reflex is retained,

and the eyelids may be seen to flutter. The tongue is very rarely bitten while the lips may be. The feces and urine are not lost. It is followed by a delirium like an anxious terror, which is as like to follow a mild, as a severe attack. There are modified and partial attacks, sometimes vertiginous, succeeded by lethargy. Globus is a sensation of a ball traveling up the esophagus, when in typical form, and is, by some, attributed to spasmodic and systematized contracture of the esophageal muscles, and by others said to be without known physiological basis.

Epileptoid Attacks, having only one or two phases of the grand attack just described, are more common phenomena, and in some cases are very difficult to differentiate from epilepsy. Attacks of tetany, of ecstasy, of sleep, or somnambulism, of noctambulism, or of catalepsy, may follow the epileptoid attacks. In catalepsy fixed positions are taken and maintained, as if the limbs were of wax. The heart and respiration are slow and weakened (Cheyne-Stokes respiration may be present), temperature is normal, or slightly subnormal; sensibility and reflexes are lost; we may have involuntary movements from the bladder and rectum, but the corneal reflex is almost always present. These attacks may last from hours to months.

In addition to these somewhat systematized phenomena, there is a variety of lesser manifestations of the hysterical state. Aphonia, which remits, comes on from emotional excitement, passes away spontaneously, or from some agent similar to that which has caused it, is a very common symptom. There is a form called aphonia spastica, which may be so sudden and severe as to demand tracheotomy. In this condition, the vocal cords are only partially movable, but it is diagnostic that the impairment is never unilateral.

Mutism. This is rare. It is diagnostic, that while the person understands everything, he is more mute than aphasic. There may be all sorts of sounds in the larynx; the person may talk backwards, and have all sorts of perversions of speech. There is a form of dyspnoea, which occurs in hysteria, which comes from a sudden laryngeal spasm, or spasm of the diaphragm, or there may be a simple increase in the rate of respiration—from 60 up to 100. Sometimes there are digestive losses. Pulmonary congestion and hemoptysis are not rare; anuria is very common, cases have been quoted where there has been no urine passed in eleven days; the cause of this condition is not certain, but has been attributed to spasm of the renal vessels. A rise of temperature is a common incident. There are two types: one where there is a continuous fever, and another, where there are temporary exacerbations of temperature. The temperature may differ upon the two sides of the body. Dissociation of pulse and temperature is common; with a pulse of 90, temperatures of 112 to 118 have

been reported. Trophic and vasomotor accidents are shown in the production of bullae, and other eruptions, in sudamina, and bloody sweat. Painful swelling, and those characterized by only apparent increase in tissue, without sensory symptoms, have been noted by many observers, of which hysterical breast is a type of one variety, and phantom tumor of another.

DIAGNOSIS. On the positive side, we may consider a case to be hysterical, if we find, in combination with symptoms of apparently organic origin, the various stigmata which have been described in the preceding pages; or if the disability in question has not the history, or distribution, or order of evolution which we have a right to expect in such conditions. On the negative side, optic neuritis, or atrophy, loss of knee-jerk or Achilles-jerk are always of organic origin. So are decided nystagmus, hemianopsia, Babinski's reflex, sustained or prominent ankle-clonus, motor or sensory paralysis in a single nerve or nerve root, incontinence of urine, except a temporary loss in spasm, and muscular atrophy with the reaction of degeneration. The mental state of hysteria is never diagnostic. A new pupillary test has been presented to differentiate between organic and psychic pain. If the pupil is contracted by the presence of a strong illumination, pressure upon an organically painful area will result in a relaxation of the pupillary ring, but will not result if the pain is of psychic origin.

TREATMENT. The treatment of this disease is not a matter for specific medication, but for management, the elimination of the causative and perpetuating conditions. If injured feelings, or shattered affections, acting upon a neuropathic foundation, are the cause, time will be a powerful factor in the therapeutics. Malnutrition is usually very evident, and the repair of the body will be followed by the return of the mind to the normal. If the disease occurs in a robust well-nourished patient, change of scene may be sufficient to work a cure. The treatment, however, in the two types of cases, does not differ so widely as might at first appear, for change of scene does not necessarily mean travel, nor can malnutrition be combatted only in a sanatorium. The patient is always courting attention, and has a morbid craving for sympathy, and a removal is often a *sine qua non* for recovery. She, for in 99 cases out of a 100 the patient is a woman, has been brought up to be idle, and self-indulged, and we ask a great deal if we are to expect the indulgent parents to stand by and assist us in what seems to be harsh and unreasonable treatment. Therefore, removal from the family, if only to another street in the same town, is often necessary. These patients are always primarily exhausted, or become exhausted by the violence of their emotions, and therefore they should be put to bed, and kept in seclusion; the bed rests them, and the seclusion eliminates the spectacular elements of the disease; but, if put in bed, they must have

massage, to get the value of passive exercise, and to equalize the peripheral circulation. They should be fed liberally, and frequently; by the nasal tube, if they will not take food otherwise. General faradism is a powerful adjuvant, both for effect upon the circulation, and for its suggestive value, and it should be given strong enough, so that the patient will be glad when the treatment is over. Cold sponge baths, alternate hot and cold sponging of the spine, are all valuable, and should be modified according to the class of symptoms presented. If the patient sleeps poorly, a bath of 100° for ten minutes at bedtime, or the hot pack, may be of assistance. The combination and manner of application of these elements in the treatment are as much a matter of good judgment, as the selection of the elements themselves. Our aim should be so to fill up the day, that the coming of night will be welcomed, and yet neither mind nor body should be exhausted in the process. A plan like this is often feasible:

7:30 A.M. Patient, while still in bed, should have a cup of cocoa.

8:00 A.M. Rise, and take a cold sponge over the whole body, standing in warm water to the ankles, if the shock is too great.

8:30 A.M. Breakfast in bed, and if the case is one of malnutrition, the nurse feeds the patient.

9:00 A.M. The patient sits in a chair, preferably in another room, while the bedroom is being cleaned for the day, and, during this time, may be read to from paper or book, or indulged in conversation.

10:00 A.M. Massage and electricity for one hour, then a cup of hot milk or broth.

11:00 A.M. to 12:00 M. Room dark, and patient in bed, and alone, but under supervision.

1:00 P.M. Dinner.

1:30 to 4:00 P.M. Conversation, a little reading, the patient may walk about the room a little, after the first week. Cup of hot milk, or broth.

4:00 P.M. Nap until 5:30, when she is dressed, talks and amuses herself until supper-time.

6:30 P.M. Supper.

7:00 P.M. Conversation until 8:00.

8:00 P.M. Gentle massage, followed by an alcohol rub, warm bath or hot pack according to circumstances.

9:00 P.M. In bed for the night.

During this period, no letters are to be written or received; no visitors, and only the most brief calls, at long intervals, from members of the family.

One great object is occupation for every moment, and after the first physical and mental protest, it promotes physical quietude and mental equanimity. As the patient grows better, the nurse

should take up with her a regular system of calisthenics, and when still better, she should begin to walk in the open air, irrespective of weather or season. Just as a pair of scales is of great assistance in stimulating the patient to increase her physique, so a pedometer will stimulate her to walk increasing distances, until she will easily cover from 8 to 10 miles a day. If exercise has a bad effect upon the symptoms, it must be reduced temporarily, and if the walking runs up the pulse or the temperature, it must be discontinued for a day or two, and then recommenced. The patient generally becomes interested in her physical development, and *pari passu* drops her mental vagaries. Healthy, physical tire promotes healthy sleep, and a proper action of the bowels, which later must be the subject of most careful attention, since intestinal toxemia perpetuates, and increases the trouble. In regard to this function, a little time spent at stool, with a good deal of massage and electricity are more beneficial than cathartics. The ingestion of a large amount of water is uniformly beneficial.

Electrical Treatment. King doubts the purely suggestive effect, and traces many failures to reliance upon this, leading to the ignoring of the proper technique. The static bombardment may do good, it is true, but, while hysterical aphonia may be instantly relieved by the sharp pain of electrical shock, it is oftener cured by passing a current through the larynx from side to side. The best treatment is to obtain the catalytic, and nutritive effects of the current upon the brain and spinal cord, as well as upon the diseased part, or member. Pass a galvanic current longitudinally and transversely through the head, then through the cord, in the same way. For motor losses, the faradic current is the better, but for sensory changes, the static spray is usually preferable, yet galvanism may act when both the other currents fail. A hyperesthesia should be treated with a soothing current, and anesthesia with a strong static spark, or faradic brush. Neuralgia of this kind is usually to be treated with the galvanic current, but may be better relieved by the faradic. Two diagnostic points will thus be elicited. If a strong faradic current is not felt over a patch of anesthesia, the cause is an organic one, and not hysterical at all. While either pole of the galvanic battery is equally potent to relieve a hysterical neuralgia, it is not true, if it has an organic basis, since in that case the anode relieves, the cathode aggravates. For clonic spasms, place the positive pole on the spasmodic muscle, current gradually raised, and as gradually decreased. This applies also to paralysis of the viscera. In hystero-epilepsy, the convulsion may be stopped by energetic electrical treatment, the variety of current not being important. The chief value of electricity in hysteria, however, is to build up the patient between attacks, rather than to treat

attacks, and this is to be done by galvanism through the head and along the spine, general faradization, or the static breeze. Treatment should be given daily. Hysterical contractures have been relieved by the static spark, and elevation of temperature, salivation, and polyuria, may be relieved by the same general treatment. As a rule the treatment must be symptomatic, aiming at the removal of a hemianesthesia, paralysis or contracture, localized neuralgic pain, asthma, globus hystericus, etc. Use such a method as will give the electrical result which you desire, and also strongly impress the patient, and therefore the faradic brush, and static spray or spark are preferable to galvanism.

THERAPEUTICS. Although no remedy can be expected to cure this disease, it may yet be the essential third factor to make efficient the hygienic, and suggestive measures, which are a necessary antecedent. We are forced to give drugs in the convulsive attacks, but the most favorable time for therapeutic action is between these attacks, or in their absence. If we can control the convulsions by our drugs, we go a great deal further than relieving an uncomfortable condition, but, owing to the conditions surrounding these cases, selection and administration are often unsatisfactory. In such a protean disease, almost any drug may be demanded at some phase, but the following are most useful.

Ignatia presents all the fickleness of hysteria in its symptomatology, and the patient is sad, depressed and indifferent, and yet wonderfully impressionable, and as intolerant of contradiction as the one for whom we prescribe *Chamomilla*. The patient is given to brooding over the actions of others, and is introspective. The motor system is as much the seat of disorder as is the mental, and there is incoördination of movement, and of the functions of the body. There is palpitation from this cause. The sensory side of the nervous system is in an irritated condition, and there is great physical sensitiveness, and a propensity to spasms, and twitchings of muscles. Paralyzes may be helped by this remedy, since they are the result of auto-suggestion, and not of organic changes.

Tarantula Hispan. In *Ignatia*, we were speaking of a remedy which acts upon the whole nervous system, while *Tarantula* affects the mind far more exclusively. The patient is a malingerer, and is full of cunning. All the limbs are in constant motion, and there is a great wildness of movement. Jousset considered the remedy to be of as much value as *Ignatia*, especially if convulsions were present. The patient appeals for sympathy constantly, while the *Ignatia* patient lives in a world of her own, and only resents intrusion. The *Tarantula* patient indulges in unusual forms of spasm, if she cannot attract attention in other ways.

Moschus is useful in cases where vasomotor symptoms are greatly in evidence. There are fainting, syncope, and globus hystericus, and the symptoms are often carried to the point of

apparent suffocation. In a recent case, the patient, a girl of seventeen, held her breath until suffocation seemed so imminent, that artificial respiration was repeatedly instituted. This drug acts best in the lower potencies, and is efficient by olefaction.

Kreasote has its principal sphere in those cases which are marked by very obstinate vomiting.

Silicea has the same sensitiveness to the opinion of others that was noted in the symptomatology of Ignatia, but is more actively whimsical and fault-finding. There are great irritability and motor restlessness, and it is particularly indicated for cases which have arisen from a shock, or exhaustion of the nervous system.

Chamomilla should be selected on its well-known mental symptoms, and is especially indicated by the persistence of joint-pains (Brodie's joints), which are sometimes the characteristic of a case.

Argentum nit. for a more chronic joint condition.

Convulsions, as a symptom, may be controlled by any of the foregoing remedies, but may need more specific treatment.

Belladonna is indicated when a strong and vigorous patient develops wild spasms and convulsions, with a red face, staring eyes, pulsating carotids, and other evidences of vascular excitement.

Stramonium for those more nearly simulating an epilepsy, with foaming at the mouth, and a fierce and maniacal condition of mind.

Hyoscyamus is more hallucinatory than the previous remedy, and the convulsions may be of a milder type.

These remedies may all fail in the convulsive attacks from the difficulty of selection, or of administration, and in such cases repeated douchings with cold water are often successful. One of the most certain measures is the hypodermatic administration of Apomorphine hydrochlorate, in doses of from one twentieth to one tenth of a grain. A single dose of this will usually bring a convulsion to an abrupt and ignoble ending in an attack of vomiting. The knowledge that a repetition of the convulsion will entail another attack of vomiting is a powerful deterrent to future exhibitions, since it is one thing to be an object of sympathy, but quite another to be ridiculous. Between attacks, an attempt should be made to meet the particular deviations from health which are presented by the particular patient. In addition to the remedies referred to above, there is frequent use for Lillium tig, Sepia, Platinum, Pulsatilla, Valeriana, Nux moschata, Lobelia inflata, Asafetida and Sulphur. Gelsemium will sometimes meet almost all the indications, in the case where vascular depression, instead of exaltation, is a prominent characteristic.

NEURASTHENIA.

DEFINITION. This is a condition of irritable weakness of both mind and body, and as a result there is an inordinate response to external stimuli, and an amount of subsequent fatigue out of proportion to the exertion, whether it has been mental or physical.

DIFFERENTIAL DIAGNOSIS. It must be differentiated from Anemia, Chronic Alcoholism, Exophthalmic Goitre, Hypochondriasis, Hysteria, Lithemia, Epilepsy of the Petit Mal type, General Paresis, Disseminated Sclerosis, Cerebral Tumor, Cerebral or Cerebro-spinal Syphilis.

AGE. No age is exempt, but young adults furnish most of the patients.

SEX. Pure forms are more common in the male.

ETIOLOGY. Heredity is the most important factor, and alcoholism in the parent is one of the most common causes of such a neuropathic constitution. Another frequent cause is tuberculosis in a parent, insanity in another, and an anemic condition of the mother at the time of pregnancy, either from malnutrition, or from too rapid child-bearing. A sedentary life with a monotonous occupation of a mental kind, involving long periods of continuous application, or general malnutrition from hurried, or scanty, or imperfectly cooked meals may give rise to it. Defective teeth may produce the condition by the imperfection of mastication, or from the absorption of the toxins from the dental disease. Occupations involving night-work, since the sleep in the day is liable to be abbreviated in amount, and is not so restful in quality as that taken at night. The above are predisposing causes.

Exciting causes are numerous, but worry is the most common and most efficient; it is the modern straining of one's powers in the race for wealth and position. Emotional shocks, whether involving what we term fright, or whether it be impending disgrace and humiliation. Traumatism even without any discoverable lesion may cause it. Alcoholism, especially if combined with excessive sexual indulgence, and the various forms of such excess, masturbation, and interrupted coitus, are competent to produce it. Any chronic physical derangement which involves distress or discomfort may produce it in a neuropathic individual, and hence, we see it following uterine disorders, neuralgias, adenoids and eye-strain. It is often toxic from alcohol, morphine, cocaine especially, and also from lead and arsenic, and certainly sometimes from tobacco. It is very often toxemic; directly from influenza, and indirectly from carcinoma, tuberculosis and like conditions. It is thought by some to be the result of auto-intoxication, since a large proportion of cases is preceded by chronic gastro-intestinal

indigestion. The early stages of Graves' Disease, Hodgkin's Disease, Addison's Disease, and Raynaud's Disease may be attended by it. Indigestion may be a symptom proceeding from the neurasthenia, but, in such case, it will be found to bear no fixed relation to meals; it is variable in extent from day to day; it will occur at times after digestible food, and be relieved by that which it undigestible; it is always accompanied by other neurasthenic symptoms, and the attacks are not amenable to the ordinary remedies for such conditions. Far more commonly we find an indigestion with a normal symptomatology. It has a history long antecedent to the neurasthenia; as this indigestion improves the neurasthenia is benefitted, and if a relapse in the gastric condition takes place the neurasthenia will be proportionately increased. If the gastric condition is chronic, we are apt to find an exacerbation of nervous symptoms at a definite, fixed period after meals, seeming to indicate that a toxic material is poured into the blood at intervals.

When we find cases of generalized disease arising from an evident intoxication, we always find the patient exhibiting headache, insomnia, restlessness (both mental and physical), nervousness, prostration, hyperesthesias, pains, irritability of temper, readiness in being startled, easy fatigue, deficient memory, and a deficiency of the power of attention. These are the symptoms also of neurasthenia, and lend great probability to the hypothesis that, however initiated, the condition actually arises from the action of toxins introduced from without, or generated within the body, generally acting in combination with a neuropathic heredity.

PATHOLOGY. While no lesions have been demonstrated, it is most probable that it is a morbid entity developed as an exhaustion of nerve-cells. Cases differ sufficiently in symptomatology to be spoken of as cerebral and spinal, but such a localization is not exact. The easy fatigue of the mind, the defective memory, and the reduction of the power of attention show that the cortical cells presiding over intellectual processes are the site of an exhaustion process. The slowness of motor response on the one hand, and the muscular jerkings and twitchings on the other, point to a weakened and irritable condition of the cells in the motor part of the cortex. These muscular starts and jerks may in other cases have their source in the pathological condition of the cells of the cord, and of the muscles themselves. These symptoms exist to some extent in almost every case, but they are not so persistent as the flushings, palpitations, bursting sensations, sometimes the actual swelling of a part in limbs and body. We know that such symptoms can result only from disturbances of circulation, and that the calibre of arteries is controlled by the sympathetic nervous system. It presides also over the secretions

and the functions of the viscera, and would explain the imperfection of digestion, and the vagaries of excretion, which are so prominent a feature of this disease. It would probably most accurately localize the lesion if we ascribed the condition to a toxic exhaustion of the general nervous system, particularly evident in the domain of the sympathetic. The reeling, swaying, dizziness, vertigo, agoraphobia, and claustrophobia, for instance, result from a disturbance of the circulation in the semi-circular canals, and the contiguous structures.

SYMPTOMATOLOGY. The symptoms may be summarized thus. Easy fatigue, irritable weakness, depression, indefinite fear, indecision, disinclination to mental or physical exertion, mental capacity unimpaired in quality, but easy fatigue makes it inefficient. Headache, vertigo, insomnia, bodily restlessness, motor weakness and tremor, deep reflexes are exaggerated. The headache may be only a feeling of heaviness, or, on the contrary, of lightness or emptiness of the head, but typically it is a splitting weight upon the brain, with a tightness of the scalp, which has been so often found that Charcot termed it the "casque neurasthenique." The neurasthenic source of this pain may be recognized from the fact that it increases on attention, and is localized according to the conception of the supposed causative disease. Hemisrania may occur. Vertigo is usually without the motor incoördination which might be expected if it were organic. The patients protest that they will fall if they attempt to walk, but can actually walk to the extent of their muscular power.

Insomnia is usual, and is persistent, and sleep is disturbed by vivid dreams, and by muscular starts and twitchings. The patients are sleepy, and the dreams are worse in the half-awakened state in the early morning.

The Eyes show a contraction of the visual fields after use (a fatigue symptom), and one patient could not look at pictures in a gallery, since the image of the first one persisted, and blurred that of the next. *Muscae volitantes* and photophobia are frequent, and eyes develop latent muscular defects. There are ringings and buzzing noises in the ears, and a very pronounced over-sensitiveness to noises. There may occasionally be a neurotic impairment of hearing. These senses show no signs of being diseased, however, but are too easily fatigued.

Gastric symptoms are almost always present. Collections of gas are common and distressing, and there are acid eructations and distress from food, generally irrespective of its character.

Constipation is present, and the patient passes large quantities of urine. Owing to the large discharge of phosphatic salts (oxalates, Oppenheim) there is apt to be some cystitis, and occasionally there are pains at the neck of the bladder, simulating the crises of tabes.

Pains are a prominent symptom, and, after the headache, the most common site is in the spine and the limbs. The area between the shoulder-blades, and at the nape of the neck, are points of election. They may be about the waist, and one patient complained of them at the "back of the stomach." The pains in the back were formerly called "spinal irritation," and "rachialgia." Sometimes one particular spot is affected, and it is accompanied by a tenderness over the corresponding spinous processes. Fixed pain is common, but it is generally in the muscles and ligaments of the back, and is such as one suffers from sitting, or standing, for a long time in one position. There is a weariness like that resulting from unaccustomed exercise. Tenderness of the spinous processes to percussion is not a characteristic symptom. While the patient may complain of severe pain, the facial expression does not confirm the statement.

Bodily restlessness with weakness is present in every case, and it even extends to inability to be contented in any place. Examination will show that every kind of movement can be executed with precision, and that there is no atrophy nor reaction of degeneration, but movements are initiated slowly, there is little force to muscular contractions, and fatigue is rapid.

Since a certain amount of hypochondriasis always accompanies neurasthenia, the weakness suggests the advent of paralysis to the patient. We can assure them that the hypothesis is fallacious, since the weakness is never confined to one nerve area, but is always general; it is unaccompanied by atrophy, and there is no reaction of degeneration.

Tremor is usually present, and is fine (may be fibrillary), and rapid. It is exaggerated by exertion, exposure to cold, and by emotional strain. Shaking fits may occur.

The Deep Reflexes (particularly the knee-jerk) are exaggerated, and we may find a foot-clonus, but this is exhausted after six or seven oscillations, thus showing that it is not organic. Rarely the knee-jerk may be very much reduced (only from extreme muscular emaciation), but if it cannot be elicited by some of the methods of re-enforcement it is organic. The superficial reflexes are greatly exaggerated, and the exaggeration of the plantar reflex may give rise to excessive jerking of the legs, and pain in the back. There is no relation between the grade of the reflex-response, and the gravity of the disease.

Changes of Sensation in the skin are common, but while paresthesias, formication, a feeling of coldness, pruritis (particularly of the anus) with tic-like twitchings, are common, anesthesias are rare. The nearest approach to anesthesia is a feeling of coldness and deadness in the extremities which is very common. This is vasomotor. Alopecia is a common symptom.

The Circulation is always disturbed, and palpitation and

tachycardia are present in some stage of all cases. There may be bradycardia, and an interruption of the contractions. The attacks may be so severe as to simulate angina pectoris. The pulse rate may go as high as two hundred to the minute. With or without a disturbance of rhythm, the whole surface of the body may flush irregularly, mostly from emotional excitement. Bursting sensations, and actual swellings of the limbs may occur. Slight irritation of the skin may so excite it that urticaria results. Heart murmurs may occur, but are rare. There may be sweating, local or general.

Sexually the patient is weakened, and he may have spermatorrhoea, or the parts may be so erethistic that masturbation is induced. Erections are another annoying result, and patients are often impotent. This is actually a hypochondriac symptom, and often has its source rather in a doubt as to their ability, than in any physical weakness. In the sexual cases, nervous cardiac symptoms, vasomotor phenomena, and nervous dyspepsia are common.

Mentally the patient is distressed by delusions and hallucinations. He lives in an atmosphere of fear, of which dread of disease is the most common. He fears to be left alone, he fears contracted surroundings, and the sight of wide spaces terrifies him. They have a fear of the obsessions which impel them to murder or suicide. Sudden noises terrify these persons almost without exception. The mind, like the body, is easily fatigued, and there is a deficiency in spontaneous thought. Inability to make, or to hold to decisions is marked. It always tends to be tinctured with, or to shade into hypochondriasis.

COURSE. It is generally years in its evolution, although some particular strain, which brings the condition to a climax, when it is no longer negligible, is often considered to be its sole cause. It generally lasts for some years, but may be marked by remissions. It always tends toward hypochondriasis.

PROGNOSIS. Cases are generally curable, more often in the male than in the female, since the female often becomes a patient from vacuity of life, which is a condition which we cannot always remedy. With a bad heredity, the prognosis is doubtful, and also if the patient is degenerate. The stronger the patient, and the more congenial the normal life, by so much is the prognosis improved. It does not tend to termination in any one of the various forms of insanity. Severe cardiac symptoms may end in atteroma. Traumatic cases tend to recovery, but it may be delayed for years.

DIAGNOSIS. This rests upon the presence of irritable weakness, both physical and mental, an increase in reflex irritability, cardiac and circulatory disturbance, secretory perversions, and tremor, either spontaneous, or readily excitable.

DIFFERENTIAL DIAGNOSIS. Hysteria. Both this and neurasthenia may be initiated by shock, and there may be an interval of weeks between the traumatism and the outbreak. Although both patients may start at slight noises, have depression, palpitation and pain in the head, at the top, and insomnia, the history of transient and anomalous nervous attacks, intermittency of many of the symptoma, and ovarian tender spots, stamps the case as one of hysteria. Neurasthenia is marked by more or less lasting exhaustion, hysteria by the occurrence of the phenomena in paroxysms. Cases are often anomalous, and difficult of identification. Hysteria has deficient will control, and an increase of reflex excitability.

Hypochondria. Neurasthenia is an exhaustion of the nervous system, and the mind shows easy exhaustibility. In hypochondriasis there is no mental feebleness, but instead an exaggerated introspection.

Lithemia. This shows a deposit of lithates in the urine, the patient has mental hebetude instead of irritability, vasomotor phenomena are absent, as are other nervous phenomena; headache is severer, but shorter in duration, they are somnolent instead of having insomnia, but the two maladies are sometimes difficult of diagnosis.

Exophthalmic Goitre. Before the eyes protrude, or the thyroid swells, it cannot be distinguished with certainty, but if the palpitation and cardiac symptoms are very prominent, and resist treatment, exophthalmic goitre should be suspected.

Chronic Alcoholism. This has tremulousness, fleeting pains in the limbs, and debility; but the feces are distinctive, the liver is disordered, and the history should be a guide.

Senile, and Other Tremors. Should be easily differentiated.

Malarial Poisoning and Anemic Conditions. These have characteristic signs, physical symptoms and history, and the same is true of Syphilis, Latent Carcinoma, Latent Tubercle, Addison's Disease, Hodgkin's Disease, etc.

Petit Mal and Migraine. Some cases have a paroxysmal quality, reminding one of petit mal, migraine, and other paroxysmal disorders. Here the physical signs are generally wanting, but petit mal has an aura, migraine scintillating scotomata,, and both patients are well between the attacks.

TREATMENT. This condition is an end-product, and no specific is known, nor is it a case for specific therapeutics. The patient must be put into the best environment, his life regulated to the minutest particular, and his general condition improved in every possible way. Time is an essential element, and must be liberal in amount. If the case is toxic, the dyspepsia and constipation must be relieved in order to stop a constant reinfection. All processes, from mastication to defecation, should be put in the best

condition. Diet, method of taking food, its quantity and the interval between meals, should be specifically stated. Enteroptosis should not be forgotten, as a patient of my own, began to improve only after a proper abdominal compress had been provided. Make a thorough physical examination, since the anemia of lasting constitutional disease often masquerades as neurasthenia. Galvanization of the cervical sympathetics and along the pneumogastrics frequently helps digestion. One of the best methods of freeing the bowels is by means of large enemata of warm water to which a little boric acid has been added. The best soap-suds injection is made extemporaneously by mixing two tablespoonfuls of soda bicarbonate with the same quantity of olive oil in a little water, and administer to the patient while lying upon the back.

Rest is Next in Importance. Use rest, but do not bar cheerful society. Isolation is not needed as it is in hysteria. Hypnotics for sleep: 12 hours sleep in the 24 hours. Take breakfast in bed. Faradism general and the D'Arsonval high-frequency current. For the nervous irritability use hot or Turkish baths. Spinal weakness will be benefitted by cold douches to the spine. It needs several weeks of baths to obtain their value. Gentle muscular exercise is good. It benefits cerebral neurasthenics, but hurts the spinal cases. A sea voyage is good "per se," and it also removes one from the possibility of injury from the routine of daily life. The moderate use of tobacco may soothe the unusual patient. One cerebral case characterized by restlessness, was observed to surreptitiously inhale the smoke from her husband's after-dinner cigar, and was noticeably better after it. She was urged to follow his example, and under the influence of one strong cigar daily made a prompt recovery.

Electrical Treatment. In approaching this form of therapy we must keep in mind that cases fall into three pretty clearly defined classes, viz., the cerebral, the spinal, and the cerebro-spinal. In the mild cases no accurate classification is demanded for electrical treatment. They will all be benefitted by a general faradic massage for ten minutes at the termination of the usual rubbing. While local galvanic treatment through the brain may be demanded in some cases, the equalization of the general circulation by general faradization has seemed to fill all the indications in the author's practice. In these cases the static machine has been of great service. Place the patient in the circuit, with the feet upon the plate, and discharge through the hair by the use of the crown. The most noticeable effect has been a sense of mental well-being, and an improvement in the circulation of the extremities. This treatment should be maintained for from ten to twenty minutes. The spinal cases have seemed to be least amenable to treatment, but the static sparks from the spine have relieved some of them. In the cerebro-spinal cases the galvanic

treatment of the cranium should be supplemented by sparks drawn from the spine, and the static breeze applied to the spine. The insomnia should always be treated by general faradism if it is well borne, but it should be kept in mind that it disagrees with, and aggravates some cases. High-frequency currents have been extensively used in neurasthenia. They are said to be contraindicated in restlessness and insomnia, and are suitable only for the cases which are relaxed, and who complain of complete lassitude. Auto-condensation with the effluve or local glass electrode along the spine has been said to be useful in such cases. The author's personal experience has been that while auto-condensation has produced striking changes in temperature and pulse rate, it has not produced much effect upon the neurasthenic condition. On the other hand if the effluve, or glass electrode, has been at the same time applied to the organ, or part, most distinctly the seat of the symptoms, the function of these parts has been very appreciably benefitted. The liver and kidneys are especially improved, but treatments applied to the spine for the relief of pains, either local, or in the distribution of the nerves from definite segments, have not been of any appreciable benefit.

THERAPEUTICS. *Abies nigra*. For atonic stomach, with depression, and mental dulness.

Artemesia absinthin. Nervous, restless weakness with vertigo.

Aconite cammarum. Erections and nocturnal emissions, without voluptuous dreams. Eructations and inclination to vomit.

Aethusa cynapium. Inability to think or to fix the attention. Stomach and abdominal pains are distinguished by their violence. Irritability, especially in the open air.

Alumina. Restlessness and mental exaltation with general fatigue, and a constipation characterized by an inability to pass even a soft stool. Must be forced out.

Ambra grisea. Nervousness and bashfulness with forgetfulness, easy fainting, and a voluptuous itching of the pudenda. Pelvic tenderness.

Ammonium inur. Coldness and sensitiveness to cold, with a fear of darkness. The head feels full, with a weight on the forehead.

Ammonium picrate. Congestive symptoms in brain and cord. Heavy feelings in occiput and mastoid region. Wild feeling in occiput following traumatism.

Amylinum nit. Flushings of face, and bursting feelings over chest. Constriction of heart and throat.

Anacardium orientale. Nerve exhaustion and nervousness, fixed ideas, obsessions, girdle sensations. Sense of complete exhaustion.

Anhalonium Lewinii. This has symptoms of mental hebetude,

tired feelings in the brain, as well as the muscles; vertigo and faintness; *muscae volitantes*, and visual hallucinations, due to a combination of cerebral and ocular disturbance. All the senses are very acute.

Argentum nit. Headache and dyspeptic symptoms from mental overwork.

Arsenicum alb. Restless weakness with irritability. Pronounced gastric irritability.

Asafetida. Great flatulence with watery stools. Great sensitiveness to physical contact, and the pains are characterized by numbness of the affected parts.

Asarum Eur. Exceeding over-sensitiveness to slight noises, with nervous irritability, and a sense of levitation.

Asclepias Syriaca. Polyuria of neurasthenia. Belching of food unchanged by digestion. Vertigo, dullness, and stupidity with a sense of constriction across the forehead.

Asclepias tuberosa. Similar, but with much vertigo.

Asterias rubens. An apprehensive state of mind with alternations of sorrow and mirth. Vertigo is prominent. There is constipation. There are nocturnal pains in the breasts, and a pushing out sensation in the uterus. The face is red and congested.

Athamantha. A confusion of the brain, indigestion, and an icy coldness of the extremities.

Aurum met. Useful in young patients with Night Terrors, which are a form of neurasthenia.

Avena sativa. In doses of 5 to 15 drops of the tincture in hot water a remedy in general.

Badiaga. General tremor with palpitation.

Baryta carb. Motor and mental asthenia.

Belladonna. With characteristic symptoms, and a peculiarly burning feeling as if in the brain.

Bellis perennis. Vertigo, easy fatigue, feeling of contraction in the scalp, "casque neurasthenique."

Cactus grand. For cases with much precordial distress.

Calc. carb. Night terrors in children.

Camphora. A feeling of utter weakness, with a conspicuous coldness of the surface, and particularly of the extremities. Insomnia, and feeble action of the heart. Visions of spirits in sleep.

Camph. brom. 3x. Nervous excitability. Hysterical alternations of mood.

Carbo veg. Flatulence, debility, with vertigo, with an aversion to darkness, and fears of ghosts.

Carduus benedictus. *Muscae volitantes*, and the eyeballs feel burning and large. Blackness before the eyes at times.

Castoreum. Nervous women with much debility, and, as an accompaniment, pains and cramps in the abdomen.

Cheonanthus virg. A case is reported of the cure, by this drug, of a clergyman, who complained of continual perspiration during the summer, weakness in arms and legs, prostrated after preaching, mind ran on his sermon and kept him awake, stupid and drowsy all the time, appetite poor, and food gave him distress.

China officin. *Muscae volitantes*, weakness physical and mental, irritable and despondent.

Cobaltum. Pronounced pain in the back, frequent nocturnal emissions, with lewd dreams, with partial, or absence of erections. Impotence.

Cocculus. Characterized by an empty feeling in the head, an opening and shutting sensation, and a sense of great bodily fatigue. Irritable weakness of all the functions of the body.

Coffea cruda. Nervous over-impressionability.

Conium. Fatigue, and particularly the muscular sense of fatigue found after unaccustomed exercise.

Conium. Nocturnal emissions, and ejaculation upon slight stimulation. Girdle sensations.

Curare. Mental and physical lassitude with lancinating headache.

Cyclamen. For depressed cases with vertigo and headache.

Cypripedium. Mental irritability, and excitement from overstrain. Especially in children.

Daphne indica. Blurring of vision with constipation, and the peculiarity that the tongue is coated on one side only. Headache provoked by intellectual labor.

Datura mel. Investigate symptomatology when extreme dilatation of the pupils is a pronounced feature. Obsessions to suicide, and violence to others, with changeable disposition, and exalted ideation with vertigo.

Digitalinum. Cardiac weakness, easy fatigue, and emissions in sleep.

Dirca palustris. Vital depression, pains in the head, flatulence, palpitation from slight causes, insomnia.

Eupatorium aromatica. Nervousness, and nervous irritability, with tremors and jerkings of the limbs.

Ferrum phos. Mental hebetude, with irritation at his deficiencies, in combination with the circulatory irritability of the *Ferrum* group.

Gelsemium. Mental and physical lassitude. Polyuria, diplopia. Muscular tremor. Symptoms in the young especially. Neuralgias in various locations.

Glonoine. Blushing and flushing of the surface with tachycardia and cardiac pain, and hammering headache, intolerance of the heat of the sun. Great sensitiveness of the head to the least jar.

Gnaphalium. Sexual excitement.

Heloderma. Symptoms of cardiac compression, with a general icy coldness, intense in degree.

Hura Braziliensis. Nervous excitability and twitchings, combined with a similar degree of irritation in the gastro-intestinal sphere.

Kali arsenicosum. Morose and depressed condition of mind, and nervousness, constricted form of headache, photophobia, loss of appetite with salivation, burning throughout the whole gastro-intestinal tract, globus and dyspnea, spinal tenderness and general muscular weakness, and cramps.

Kali phos. Despondency, anxiety, fearfulness, tearfulness, homesickness, suspiciousness, agoraphobia, weak memory; small frequent pulse, and later a retarded one; pains with the sensation of paralysis; weakness of muscles even to paralysis; retarded nutrition, depression of function throughout the body.

Lachesis. This is much like the preceding, but with a predilection for the left side in all symptoms, and a remarkable dislike to being touched.

Magnesia carb. There is a typical nervous exhaustion. The keynote is sensitiveness. This relates to mental impressions, touch, cold air, shocks and blows. This hyperesthesia often passes into neuralgias.

Methylene Blue. In the 3x, neuralgias of neurasthenia, tremors and trophic disturbances; in irritations of the spinal cord, and where the patient is very tympanitic.

Onosmodium. Vertigo and general sensations of numbness, and a prostration of muscular strength. Neuralgic pains with this. Numb feelings in the head. Spinal cord irritation, and sexual excitement. Cases which have been developed from sexual excess.

Oxalic acid. Neurasthenic cases with notable cardiac symptoms, palpitation and irregular action alternating with aphonia.

Phosphoric acid. Marked apathy and drowsiness; confused and disconnected thoughts. Mental exertion makes him dizzy, vertigo with the symptom that that upon which he is sitting is rising; unequal dilatation of the pupils; muscular weakness and polyuria.

Pieric acid. Easy fatigue, both mental and physical. Mental exertion gives rise to burning pains in the spine, occipital headache. Sexual excitement, but with loss of power. A patient of the author's had emissions on mental exertion. The remedy with the static breeze, and improved hygiene relieved the condition.

Platina. Arrogance is the keynote, alternation from joy to terror, spine painful, "casque neurasthenique."

Sabal serrulata. Mental irritability, but with weakness. Sexually excited; polyuria.

Senecio aureus. Globus is a marked symptom, and it is

peculiarly, and perhaps only applicable to cases where the condition has arisen from suppressed or deficient menstruation.

Stannum. This has mental and physical weakness as its cardinal symptom. The mental weakness is directly due to cardiac weakness; it is an ischemia. This is proven by the symptom that the weakness is felt more on going down, than in ascending stairs. There is also much palpitation. There is a sinking feeling in the epigastrium, and in the chest.

Strychninum. The peculiarly constricted headache, often found in neurasthenia. Facial neuralgia, and neuralgic headache. Pains and chills in the occiput, and nape of the neck, which run down the spine. Itching over the whole body, and particularly in the roof of the mouth. Jerkings and twitchings of the muscles, and limbs, with a hypersensitiveness to all external stimuli. This last is very pronounced.

Thyroidium. For puffy and obese patients.

Vipera communis. This is almost a specific for the bursting sensations in limbs "if they are allowed to hang down."

Zincum. Cases described as "Brain Fag." It affects muscle, nerve, and brain. It applies to cases where the neurasthenia is not the expected failure of a congenital neurasthenia, but where the condition is the legitimate result of overstrain from work, or dissipation.

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1. The first part of the document is a list of names and addresses of the members of the committee.

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